H. H. Hickman

British Journal of Anaesthesia

JOINT EDITORS:
E. FALKNER HILL T. CECIL GRAY
Editor of Postgraduate Educational Numbers R. P. HARBORD

Volume XXX No. 2 February 1958

Clendening Medical Library
APR 7 1958
University of Kansas Medical Center

JOHN SHERRATT & SON : *Publishers*
ALTRINCHAM ENGLAND

MIE LTD

present

THE PINKERTON (INFLATABLE)

CUIRASS

Described by Dr. H. H. Pinkerton, M.B., Ch.B., F.R.F.P.S.G., F.F.A.R.C.S., Western Infirmary, Glasgow.
(Ref. Brit. J. Anaesth. (1957), 29, 421.)

For the alternate inflation and deflation of the chest providing adequate pulmonary ventilation during Laryngoscopy and Bronchoscopy under general anaesthesia

FULL DETAILS ON REQUEST

MEDICAL & INDUSTRIAL EQUIPMENT LTD.

SPECIALISTS IN ANAESTHETIC AND RESPIRATORY APPARATUS

10 & 12 NEW CAVENDISH STREET, LONDON W.1

Telephone : WELbeck 1851 and 1504 *Telegrams : NARCOSIS, LONDON*

MANCHESTER: 152-154 Oxford Road
Phone : ARDwick 5906

TORONTO, CANADA: 83-85 Grenville Street
Phone : WALNUT 3-3845

"*I would have everie man write what he knowes and no more.*"—MONTAIGNE

BRITISH JOURNAL OF ANAESTHESIA

VOL. XXX, No. 2 FEBRUARY 1958

EDITORIAL

DEATH ASSOCIATED WITH ANAESTHESIA

DURING the past eight years or so, anaesthetic deaths have been the subject of much investigation. A committee set up to study such deaths issued in 1956 a comprehensive report based on their review of 1,000 records of deaths associated with anaesthesia. From this report a vast amount of valuable clinical information has been made available to anaesthetists.

Little, however, has previously been written concerning the medico-legal aspect of deaths associated with anaesthesia and in this issue Dr. Lawrie examines in detail the procedure involved in the reporting and registration of such deaths as it applies in Scotland. Dr. Lawrie criticizes the reporting of these cases in that so much emphasis is placed on the anaesthetic that it is almost inevitable that the anaesthetic administered will appear on the death certificate as at least a contributory cause of death, whereas, in many cases, it represents merely an incident in the patient's surgical treatment and, in fact, may have been recovered from prior to death. Many anaesthetists in Scotland are in agreement with Dr. Lawrie's views on this subject and feel that some modifications in the existing procedure would be timely.

Firstly, it would obviously be of great value if an acceptable definition of "Death Associated with Anaesthesia" could be agreed upon between anaesthetists and the appropriate legal authority. This admittedly would be no easy task as in some areas anaesthetists are required to report all cases which die within twenty-four hours of operation, while in other areas an assurance from the surgical team that the patient had in fact fully recovered from the anaesthetic before death will be accepted.

Secondly, the form on which such deaths are reported might be redesigned to include details of the surgical procedure as well as those concerning the anaesthetic. This form could be completed jointly by surgeon and anaesthetist and with the additional information available, and should allow of a more critical evaluation of the relative effects of anaesthesia and surgery in determining the cause of death.

It is stressed that these suggestions are not intended to limit in any way the statutory investigation of deaths occurring during or shortly after anaesthesia but are put forward in support of Dr. Lawrie's plea that the inclusion of anaesthesia as a contributory cause of death should be reserved for those cases where it can be fairly adjudged to have played a part.

Brit. J. Anaesth. (1958), **30**, 50

THIOPENTONE INDUCTION AND THE ELECTROCARDIOGRAM

BY

W. N. ROLLASON AND J. M. HOUGH

Royal Infirmary, Hull, England

SINCE 1932 the thiobarbiturates have been extensively used intravenously as a pleasant and effective means of inducing anaesthesia. Of these thiopentone is the most popular.

The effect of this drug on the heart depends on the pre-operative state of the cardiovascular system and on the rate and amount of the drug injected. It is reputed to weaken the force of the heart's contraction, to cause it to dilate and to depress the blood pressure by dilatation of the vascular bed.

Some investigators (Volpitto and Marangoni, 1938; Ruth et al., 1939; Hale, 1954) have commented on the absence of e.c.g. changes following induction of anaesthesia with thiopentone, while others (Betlach, 1938; Carraway, 1939; Johnstone, 1951; Harrison, 1953; Stephen et al., 1953; Rollason and Cumming, 1956*) have reported changes.

This paper is based on a series of 52 unselected cases in which all components of the e.c.g. were investigated in detail before and after induction of anaesthesia with thiopentone using both standard and chest leads connected to an Elmquist Triplex Machine.

MATERIAL AND METHODS

Of the 52 cases 26 were females and 26 were males. Their ages ranged from 18 to 71 years and their weights from 6 st. 6 lb. (41 kg) to 14 st. 4 lb. (91 kg).

Four patients were hypertensive (case nos. 28, 31, 34, 43; table I). All cases had a normal haemoglobin concentration and the blood urea was within normal limits in all except one (case no. 28) whose blood urea was 44 mg per cent.

All had normal teleradiograms except two, one (case no. 28) showing gross and the other (case no. 45) showing moderate cardiomegaly.

Premedication varied as shown:

Premedication	No. of patients
Amidone + Atropine	2
Pethidine + Atropine	8
Pethidine + Scopolamine	4
Papaveretum + Scopolamine	16
Pethidine + Scopolamine + Chlorpromazine	12
Pethidine + Promethazine + Chlorpromazine	5
Papaveretum + Scopolamine + Chlorpromazine	5
Total	52

The induction dose of thiopentone was injected slowly and ranged from 2.9 to 8.6 mg per kg, the average being 5.9 mg/kg. The dose of thiopentone was below the average in 18 of the 22 cases receiving chlorpromazine as part of their premedication.

Standard leads and chest leads V1, V3 and V5 e.c.g. tracings were taken before and two minutes after the induction dose of thiopentone.

A clear airway was maintained and respiration was not significantly depressed. No patient was inflated with oxygen and each breathed atmospheric air.

In addition to changes in blood pressure and pulse rate, changes in the various components of the e.c.g. were also sought. These included changes in the P wave, PQ, QRS and QT intervals, QRS voltage, axis rotation and ST segment and T wave changes.

RESULTS

In 49 cases the systolic blood pressure fell, the average fall was 26 mm Hg and the average fall in diastolic pressure was 10.5 mm Hg.

In 3 cases (nos. 10, 14, 22) the systolic pressures rose, the average rise being 8.3 mm Hg, and in

* This paper was a preliminary survey of e.c.g. changes associated with hypotensive anaesthesia. Much of the same data is used in the present paper, but a more detailed measurement of the e.c.g. records has been made and a specific significance level adopted to eliminate minor changes in the e.c.g. pattern.

TABLE I

Case no.	Sex	Age	Weight (kg)	Dose of thiopentone (mg/kg)	B.P. (mm Hg)		P.R./min	
					Pre	Post	Pre	Post
1	M	57	66	6.8	140/80	120/80	120	120
2*	F	51	52	3.8	110/80	80/60	75	85
3*	F	52	46	5.4	80/50	70/50	80	80
4	M	19	74	6.7	130/80	110/75	80	95
5	F	68	85	5.3	130/90	105/80	95	95
6*	M	54	61	6.6	130/80	90/70	85	100
7*	M	28	62	4.8	105/50	100/50	100	105
8	F	54	69	7.2	160/100	140/80	65	75
9	M	53	58	8.6	120/80	100/70	85	100
10	M	55	62	4.8	95/70	100/75	60	95
11	F	19	41	7.3	145/80	120/70	135	105
12	M	30	60	8.3	120/80	105/70	60	75
13	F	41	55	7.3	160/105	110/80	135	120
14	M	59	91	5.5	120/85	130/90	80	120
15*	M	46	75	5.3	115/80	100/60	130	130
16*	M	59	85	4.1	120/80	95/65	100	90
17*	F	31	60	4.2	100/65	90/60	95	100
18	F	46	71	7.0	170/90	135/85	75	85
19*	F	34	64	4.7	120/70	100/60	150	120
20*	F	47	48	8.3	120/65	85/60	75	95
21*	F	47	61	4.1	130/70	115/65	105	115
22*	M	26	70	3.6	120/75	130/75	110	125
23	M	41	62	8.1	105/75	80/60	80	105
24*	F	51	46	4.3	120/70	90/55	150	150
25	F	65	56	4.5	130/80	75/50	95	100
26	M	53	70	5.7	105/65	85/65	75	105
27	M	21	60	8.3	105/60	85/50	65	85
28*†‡	M	42	66	4.5	270/160	230/120	120	115
29*	F	38	62	3.2	110/70	90/65	115	110
30	F	45	68	6.6	130/80	90/60	105	105
31†	F	51	54	7.4	200/105	120/80	65	85
32	F	41	77	6.5	140/90	100/80	95	105
33	F	38	50	6.0	140/80	100/75	100	120
34*†	F	58	60	3.3	185/100	150/100	85	100
35	M	47	61	4.9	100/65	85/60	55	65
36	M	45	71	7.0	110/70	90/65	75	105
37	M	28	68	7.3	125/85	100/80	60	120
38*	M	40	70	2.9	115/65	95/65	120	120
39*	M	31	59	5.1	120/70	115/65	100	120
40*	F	33	49	3.6	115/65	95/50	95	85
41*	M	44	72	6.9	80/60	70/50	65	85
42	M	47	62	8.1	120/85	90/65	95	120
43†	M	62	68	4.4	170/105	130/100	120	105
44	F	57	70	7.1	130/80	90/70	105	105
45‡	F	71	51	5.9	140/80	95/60	120	105
46	F	46	73	6.8	140/80	90/65	100	120
47	M	53	63	7.1	115/75	100/60	85	95
48	F	63	62	5.6	130/70	110/80	105	120
49	M	48	59	8.5	150/75	100/55	85	100
50*	F	56	81	3.7	120/90	105/75	85	90
51*	F	65	41	4.9	120/75	100/60	85	80
52*	M	18	62	8.1	125/85	95/65	110	125

* Cases receiving chlorpromazine as part of their premedication.
† Hypertensive cases. ‡ Cases with cardiomegaly.

TABLE II

Alteration in P.R.	No. of cases	Before thiopentone induction		After thiopentone induction	
		P.R. range	Average	P.R. range	Average
Increase	34	55–110	81.6	65–125	99.3
Decrease	10	85–150	117.5	80–120	103.5
No change	8	80–150	113.1	80–150	113.1

TABLE III
P wave changes

Case no.	Lead	Height before thiopentone induction in mm	Height after thiopentone induction in mm	Change in mm
4	II	1.5	3.0	+1.5
	III	0..5	1.5	+1.0
18	II	2.0	3.5	+1.5
26	II	1.0	3.0	+2.0
	III	0.5	2.0	+1.5
33	III	1.0	2.0	+1.0
36	II	0	1.5	+1.5
	III	0	1.0	+1.0
47	II	1.0	2.0	+1.0

Significance level = 1 mm.

Case no. 36 is illustrated in figure 1.

3 cases (nos. 10, 14, 48) the diastolic pressure rose, the average rise being 6.7 mm Hg.

Changes in pulse rate are illustrated in table II, from which it will be seen there was an average increase in pulse rate of 17.7 per minute in 34 cases, an average decrease of 14 per minute in 10 cases and no change in 8 cases.

Six cases showed an increase in the height of the P wave after thiopentone induction. These are illustrated in table III.

Cases showing a change of less than 1 mm were not recorded. The change was present in lead II in 5 cases and in the lead III in 4 cases. The maximum change observed was 2.0 mm and this was recorded in lead II (case no. 26).

In one case (no. 38) notching of the P wave became evident after induction with thiopentone.

There were no cases showing changes in the PQ and QRS intervals but cases showing changes in the QTc interval (i.e. QT interval corrected for pulse rate) are illustrated in table IV. Changes of less than 0.04 sec were not recorded. It will be seen that there was a prolongation of the QTc interval after thiopentone induction in 16 cases, the prolongation ranging from 0.04 sec to 0.15 sec and a shortening of the interval in 2 cases, the shortening being only 0.04 sec in each case.

TABLE IV
Changes in the QTc intervals in seconds.

Case no.	Before induction	After induction	Change
12	0.38	0.42	+0.04
16	0.28	0.37	+0.09
19	0.35	0.39	+0.04
20	0.40	0.48	+0.08
23	0.35	0.40	+0.05
27	0.29	0.33	+0.04
33	0.44	0.48	+0.04
34	0.38	0.44	+0.06
37	0.32	0.45	+0.13
39	0.33	0.37	+0.04
41	0.41	0.47	+0.06
42	0.40	0.45	+0.05
43	0.45	0.53	+0.08
45	0.39	0.45	+0.06
46	0.46	0.42	−0.04
48	0.35	0.40	+0.05
49	0.40	0.36	−0.04
52	0.30	0.45	+0.15

Significance level = 0.04 sec.
Case no. 37 is illustrated in figure 4 and case no. 45 is illustrated in figure 3.

Cases showing changes in the electric axis and in the QRS electric vector are illustrated in table V.

It will be seen that 12 cases show changes in the electric axis in excess of 50° after induction with thiopentone, the maximum being 59° (case

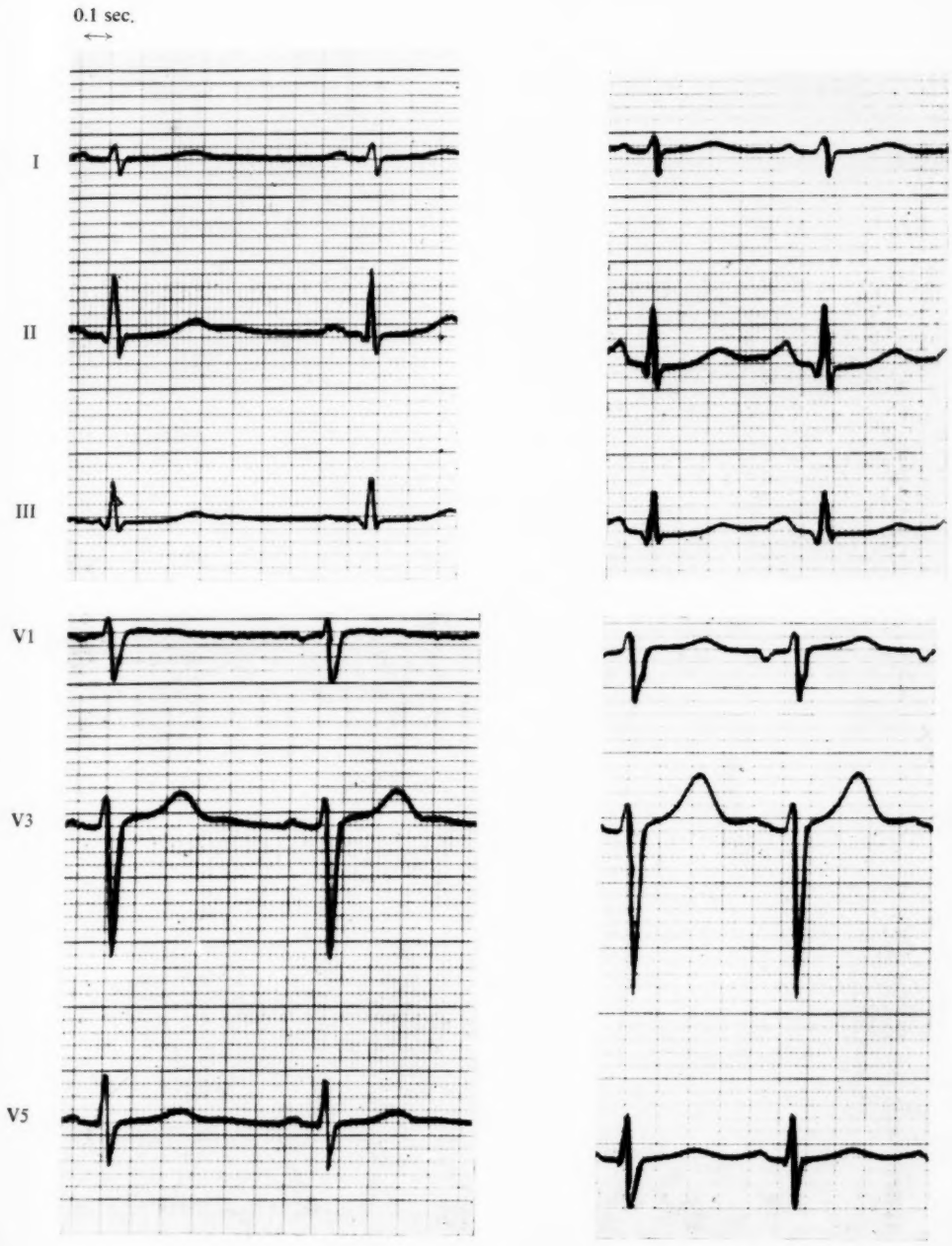

FIG. 1
Case no. 36. Changes in P2 and P3, electrical axis, and TV3.
A. Before thiopentone induction. B. After thiopentone induction.

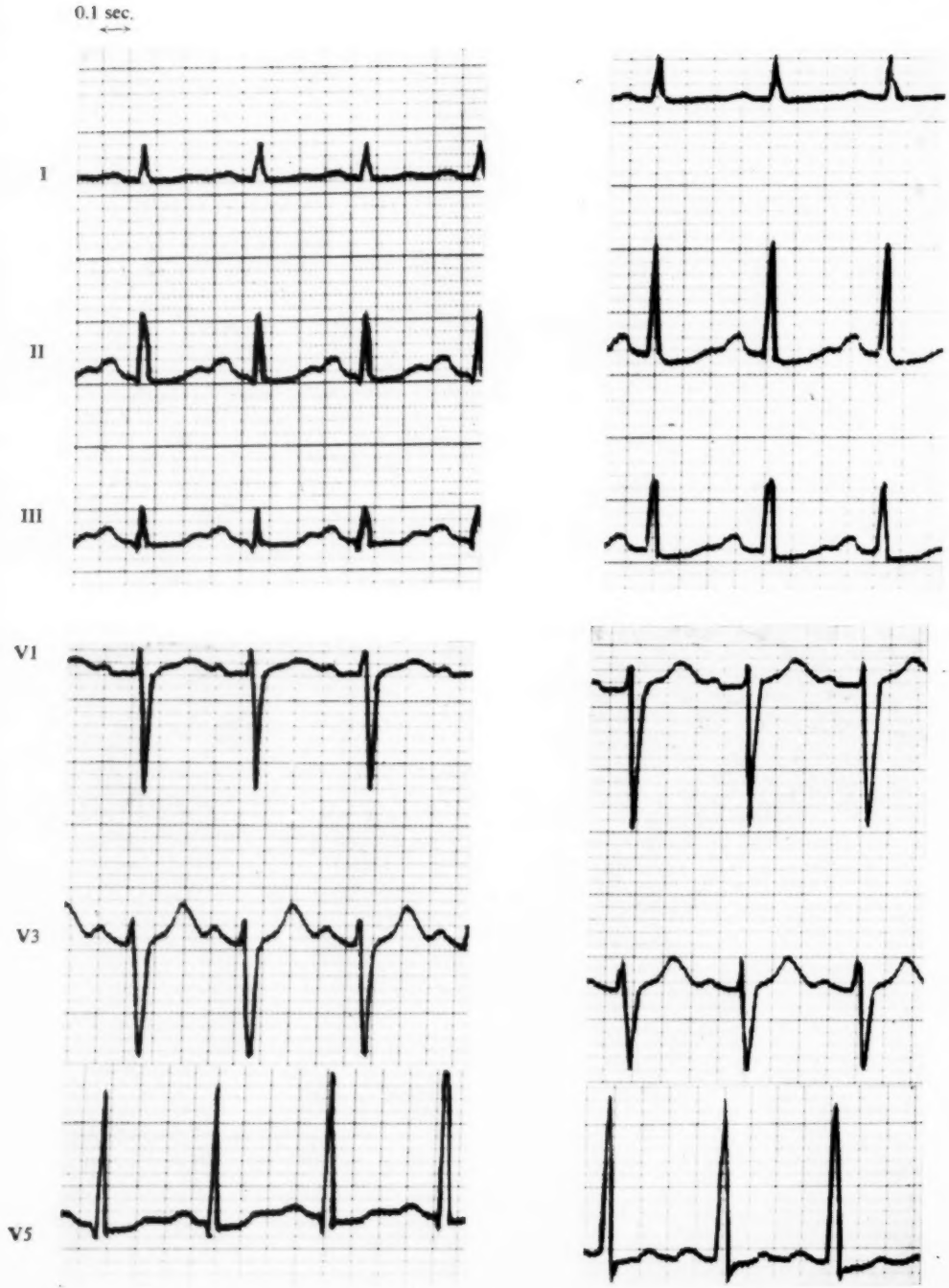

FIG. 2
Case no. 24. Changes in electric axis and QRS electric vector.
A. Before thiopentone induction. B. After thiopentone induction.

TABLE V
Change in QRS.

Case no.	Electric axis (degrees)			Electric vector (mm)			Chest leads showing changes
	Before induction	After induction	Change	Before induction	After induction	Change	
8	25	37	12	12.0	14.0	2.0	None
10	60	52	− 8	11.5	9.5	2.0	V5
11	59	67	8	11.5	15.5	4.0	V3 & V5
22	67	44	−23	9.5	8.0	−1.5	None
24	48	69	21	8.5	18.0	9.5	V1, V3 & V5
34	37	49	12	13.0	14.5	1.5	V3 and V5
35	135	76	−59	5.0	10.0	5.0	None
36	87	114	27	5.0	4.0	−1.0	V3
41	30	42	12	10.5	10.5	0	None
42	64	53	−11	16.0	16.0	0	V3
43	74	73	− 1	13.5	10.0	−3.5	V5
44	11	18	7	7.5	9.0	1.5	V3
51	−18	−18	0	8.0	12.0	4.0	None
52	30	75	45	1.5	11.5	10.0	V1 & V5

Significance level for electric axis = 5°.
Significance level for electric vector = 3.0 mm.
Case no. 24 is illustrated in figure 2 and case no. 36 is illustrated in figure 1.

No detailed analysis was made of changes in the chest leads as any change in axis rotation or electric vector in these leads must be related to similar changes in the standard leads and calculation of the axis rotation and the electric vector from the chest leads is not easily made.

The method of calculation of the electric axis and the electric vector used was as illustrated in the appendix of a previous paper (Rollason and Hough, 1957) but for those who prefer to avoid mathematics the axis can be obtained from Dieuaide's Chart (Burch and Winsor, 1949).

TABLE VI
Change in ST segment only after thiopentone induction.

Case no.	E.C.G. leads			Changes in mm		
	I	II	III	VI	V3	V5
24	0	−1.75	0	0	0	0

Case no. 24 is illustrated in figure 2.
+ = elevation.
− = depression.
0 = No change or below significance level of 1 mm in standard leads and 2 mm in chest leads.
* = Change of sign.

no. 35), and 11 cases show changes in the electric vector of 2.0 mm or more, the maximum being 10 mm (case no. 52).

One case showed a change in the ST segment only after induction with thiopentone, 2 cases showed changes in both ST segments and T waves, whereas 22 cases showed changes in the T wave only. These are illustrated in tables VI, VII, and VIII.

The effect of thiopentone induction on cardiac arrhythmia in the series is illustrated in table IX.

DISCUSSION

Pulse rate. The changes in pulse rate after thiopentone induction assumed the same general pattern as those reported by Johnstone (1951). Average pulse rates in the region of 120 per minute showed a decrease in rate after thiopentone, those in the region of 80 per minute showed an increase in rate whereas those in the region of 115 per minute showed no change.

Blood pressure. The generally accepted effect of thiopentone to depress the blood pressure was confirmed; the average fall in systolic pressure amounted to 26 mm Hg and average fall in diastolic was 10.5 mm Hg.

P wave changes. Increase in height of the P wave was observed in 6 cases. This increase ranged from 1 to 2 mm and was revealed in standard leads II and III. Increase in the amplitude of the P wave after thiopentone has also been observed by Betlach (1938) and Johnstone (1951). The height of the P wave should normally not be above 2 mm but this was exceeded in 3 instances (case nos. 4, 18, 26). In no case did the

duration of the P wave exceed the normal limit of 0.1 sec.

In this series the P wave changes were associated with an increase in pulse rate and a decrease in systolic blood pressure. They occurred in patients under 55 years of age with normal cardiorespiratory systems and were related to the higher dosage of thiopentone but were unrelated to sex or weight (table X).

No patient who received chlorpromazine as part of the premedication showed a significant P wave change.

Tall sharp P waves ranging from 2 mm to 5 mm are usually associated with right auricular hypertrophy (Wood, 1956) and presumably the high voltage P waves seen in some cases after induction with a substantial dose of thiopentone and associated with an increase in pulse rate reflect a compensatory increase in auricular activity to offset the fall in blood pressure.

A bifid P wave was evident in leads II and III in one case (no. 38) after induction with thiopentone, but there was no alteration in its height or duration, which were within normal limits.

TABLE VII

Changes in ST segments and T waves after thiopentone induction.

	E.C.G. leads				Changes in mm							
Case no.	ST I	T I	ST II	T II	ST III	TIII	STVI	TVI	STV3	TV3	STV5	TV5
43	+1.0	0	+1.0	−1.5	0	0	0	0	0	0	0	0
45	+1.25	−1.0	0	+1.0	0	0	0	0	0	+3.0*	0	0

Case no. 45 is illustrated in figure 3.

\+ = elevation.
− = depression.
0 = No change or below significance level of 1 mm in standard leads and 2 mm in chest leads.
* = Change of sign.

TABLE VIII

Changes in T wave only after thiopentone induction.

	E.C.G. leads		Changes in mm			
Case no.	I	II	III	VI	V3	V5
1	0	−1.5	0	0	0	0
2	0	0	0	+1.5*	+2.0*	0
4	0	−1.0	0	0	+2.0	0
7	+1.0	+1.5	0	−5.0*	0	0
10	0	−1.0	0	0	−3.0	−4.75
13	0	0	0	0	−2.0	0
15	−1.0	0	0	0	0	0
18	−1.5	0	0	0	0	−2.5
19	0	−1.0	0	0	0	0
21	−2.5	−1.0	0	0	0	0
23	0	0	0	0	0	−2.5
26	0	0	0	0	−3.0	0
28	0	−1.0	0	0	−3.5	0
31	0	0	0	0	−2.0	0
32	0	0	0	0	+4.0*	0
33	0	0	0	0	−2.0	0
34	−1.25	−1.5	0	0	−5.0	−5.0
36	0	0	0	0	+3.5	0
37	−2.0	−1.0	0	+3.0	+3.0	−6.0
38	0	0	0	0	−2.0	0
44	−1.0	−1.0	0	0	0	0
52	0	0	0	0	−3.0	0

It will be seen that leads II and V3 were the most and lead III the least informative. Case no 36 is illustrated in figure 1 and Case no. 37 is illustrated in figure 4.

\+ = elevation.
− = depression.
0 = No change or below significance level of 1 mm in standard leads and 2 mm in chest leads.
* = Change of sign.

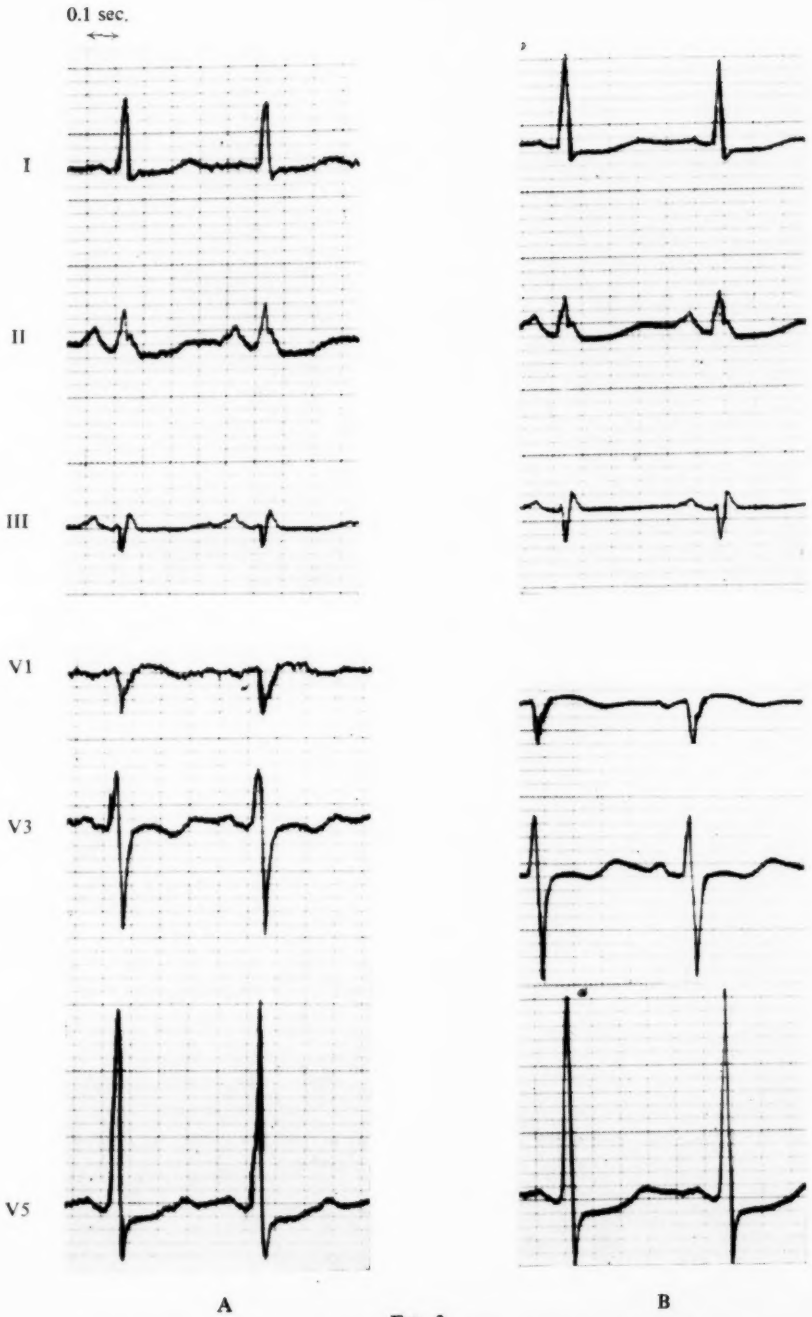

FIG. 3
Case no. 45. Changes in ST1, T1, T2, TV3, and prolongation of QTc.
A. Before thiopentone induction. B. After thiopentone induction.

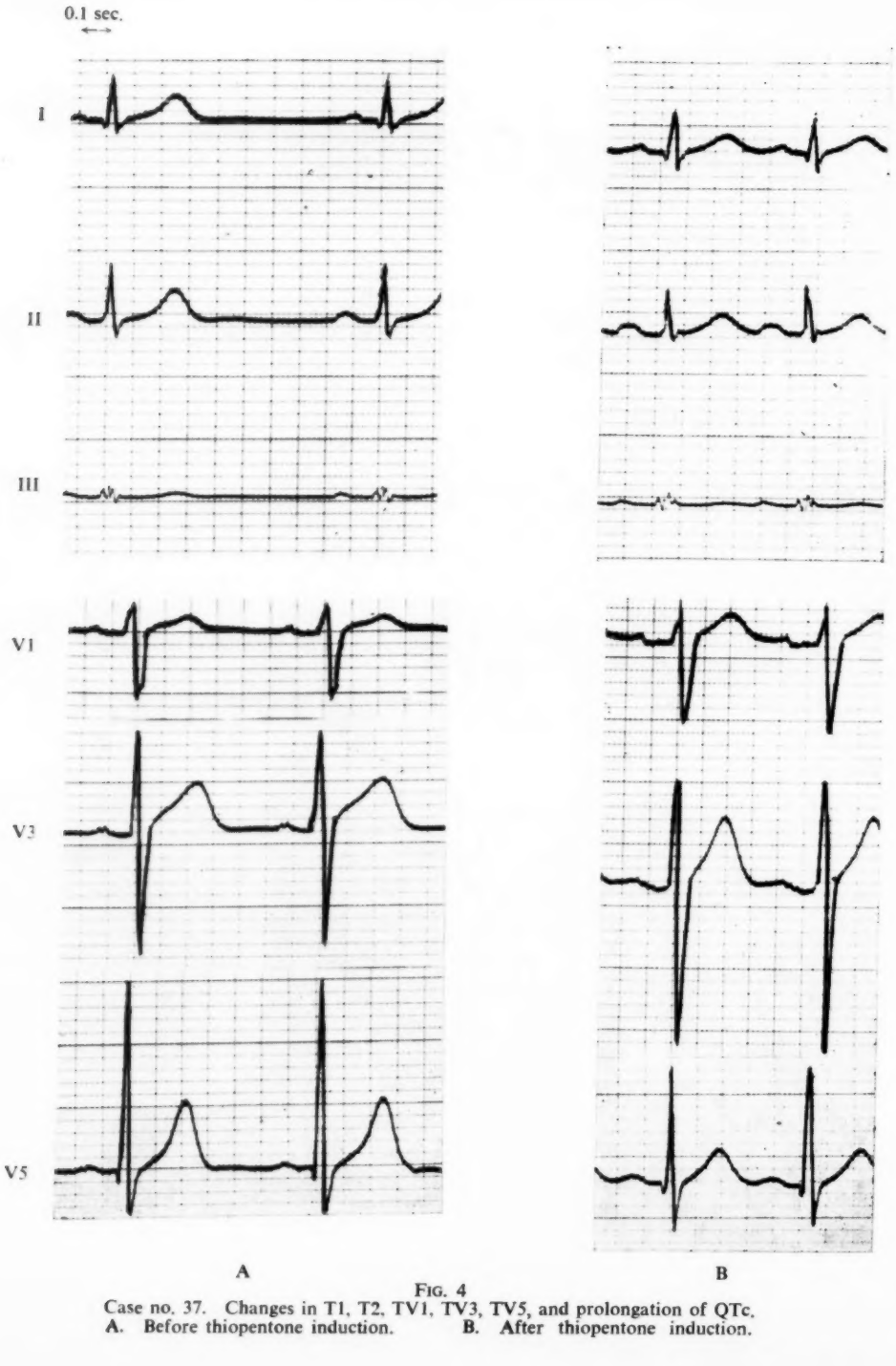

FIG. 4
Case no. 37. Changes in T1, T2, TV1, TV3, TV5, and prolongation of QTc.
A. Before thiopentone induction. B. After thiopentone induction.

No case of nodal rhythm following thiopentone induction was observed in this series.

PQ QRS and QTc intervals. There were no significant changes in either the PQ or QRS intervals and all were within normal limits.

The QTc interval was calculated with the aid of a slide rule from the actual QT and the cycle length ($QTc = \sqrt{c}$ where c = cycle length) and was found to vary more than QT itself.

The normal QT interval is inversely proportional to the heart rate ranging from 0.42 at 48 beats per minute to 0.28 at 110 beats per minute.

The QTc interval was prolonged after thiopentone induction in 16 cases and shortened in 2 but these changes could not be related to the age, sex or weight of the patients, to the premedication or dose of thiopentone or to changes in pulse rate or blood pressure. One of the two cases with cardiomegaly showed a prolongation of QTc of 0.06 sec (case no. 45) but the other (case no. 28) showed no significant change.

Of the 4 hypertensive cases (nos. 28, 31, 34, 43) 2 (nos. 34, 43) showed a prolongation of QTc but the other 2 revealed no significant change.

QTc is reputed to be lengthened by cardiac enlargement and shortened by cardiac compression, but there was no suggestion that the heart was compressed after induction with thiopentone, but it is reputed to dilate.

Axis rotation. Twelve cases showed significant changes in the electric axis after thiopentone induction and these are illustrated in table XI.

No correlation could be seen between the axis rotation and the sex, age or weight of the patients, or to the dose of thiopentone administered. Neither could any correlation be seen between the axis rotation and the alterations in blood pressure and pulse rate.

Some depression of respiration, however, follows induction with thiopentone and it is possible that changes in axis are due to the consequent variations in conduction produced by alterations in contact of the heart with the adjacent conducting media, and it is known that where these contacts vary, such as in those with long thin chests, there is a tendency to right axis deviation (positive axis rotation exceeding 90°) while in those with a high diaphragm the tendency is to left axis deviation (negative axis rotation).

QRS electric vector. Six cases showed significant changes in the QRS electric vector after thiopentone induction and those are illustrated in table XII.

The method of calculation was as illustrated in the appendix of a previous article (Rollason and Hough, 1957).

It will be seen that all the vector changes after thiopentone induction were associated with a fall in the blood pressure and 3 cases (nos. 11, 43, 51) were associated with a fall in the pulse rate, whereas all significant changes in the height of the P wave after thiopentone induction were associated with a rise in the pulse rate.

The one case (no. 43) showing a decrease in the QRS electric vector and a fall in pulse rate was associated with the largest fall in pulse pressure, i.e. 35 mm Hg. The fall in pulse pressure in those showing an increase in the electric vector ranged from 0 to 15 mm Hg.

There was no significant correlation between the vector changes and the sex, age or weight of the patients, or with the dose of thiopentone used.

Changes in the QRS electric vector can occur when there is an increased or decreased electric impulse from the heart and when there is an alteration in conductivity of the electrical impulse from the heart to the electrodes.

In those cases showing a significant change in the electric vector the general effect of thiopentone induction was to increase it by an average of 4.8 mm. As a decreased respiratory excursion follows induction with thiopentone it is possible that this results in increased conductivity from the heart to the electrodes, resulting in a rise in the vector, and in the case showing a decrease in the electric vector after thiopentone it is possible that the reduction in pulse pressure and pulse rate in this case resulted in a decrease in the electrical impulse from the heart itself greater than any associated increased conductivity in its adjacent media due to diminished respiratory excursion. In future the clinical estimate of diminished tidal volume is to be replaced by accurate measurement with a Dräger volumeter.

ST segment changes. These were significant in 3 cases (nos. 24, 43, 45) and in 2 cases (nos. 43, 45) there were significant T wave changes in addition. The ST changes which were depressions

TABLE IX

Changes in Cardiac rhythm after thiopentone induction.

Case no.	Supraventricular	Ventricular
1	No change.	Ventricular extrasystoles appeared.
5	No change.	Ventricular extrasystoles appeared.
11	Auricular extrasystoles disappeared.	No change.
24	No change.	Ventricular extrasystoles disappeared.
35	No change.	Ventricular extrasystoles disappeared.
38	No change.	Ventricular extrasystoles disappeared.
40	No change.	Ventricular extrasystoles appeared.

TABLE X

Case no.	Sex	Age	Weight (kg)	Dose of thiopentone (mg/kg)	B.P. (mm Hg)		P.R./min	
					Before	After	Before	After
4	M	19	74	6.7	130/80	110/75	80	95
18	F	46	71	7.0	170/90	135/85	75	85
26	M	53	70	5.7	105/65	85/65	75	105
33	F	38	50	6.0	140/80	100/75	100	120
35	M	47	61	4.9	100/65	85/60	55	65
47	M	53	63	7.1	115/75	100/60	85	95

Average dose for the series = 5.9 mg/kg.

TABLE XI

Case no.	Sex	Age	Weight (kg)	Dose of thiopentone (mg/kg)	B.P. (mm Hg)		P.R./min		Axis change (degrees)
					Before	After	Before	After	
8*	F	54	69	7.2	160/100	140/80	65	75	12
10*	M	55	62	4.8	95/70	100/75	60	95	8
11*	F	19	41	7.3	145/80	120/70	135	105	8
22†	M	26	70	3.6	120/75	130/75	110	125	23
24*†	F	51	46	4.3	120/70	90/55	150	150	21
34†	F	58	60	3.3	185/100	150/100	85	100	12
35*	M	47	61	4.9	100/65	85/60	55	65	59
36	M	45	71		110/70	90/65	75	105	27
41†	M	44	72	6.9	80/60	70/50	65	85	12
42	M	47	62	8.1	120/85	90/65	95	120	11
44	F	57	70	7.1	130/80	90/70	105	105	7
52†*	M	18	62	8.1	125/85	95/65	110	125	45

* Cases showing a significant change in the QRS electric vector in addition.
† Cases receiving chlorpromazine as part of their premedication.

TABLE XII

Case no.	Sex	Age	Weight (kg)	Dose of thiopentone (mg/kg)	B.P. (mm Hg)		P.R./min		Change in QRS vector (mm)*
					Before	After	Before	After	
11	F	19	41	7.3	145/80	120/70	135	105	+4.0
24†	F	51	46	4.3	120/70	90/55	150	150	+9.5
35	M	47	61	4.9	100/65	85/60	55	65	+5.0
43	M	62	68	4.4	170/105	130/100	120	105	−3.5
51†	F	65	41	4.9	120/75	100/60	85	80	+4.0
52†	M	18	62	8.1	125/85	95/65	110	125	+10.0

* Significance level = 3 mm. Average change in the QRS electric vector = +4.8 mm.
† Cases receiving chlorpromazine as part of their premedication.

were revealed in leads I and II and occurred in patients over 50 years of age.

Case no. 24 had received chlorpromazine as as part of the premedication; case no. 43 was hypertensive prior to induction with thiopentone; and case no. 45 had moderate cardiomegaly.

Each case was associated with a fall in blood pressure and either no change or a fall in pulse rate, and it is possible the ST changes following thiopentone induction were due to a reduction in coronary flow proportionately greater than the reduction in cardiac work resulting in an oxygen differential in the heart (Beck, 1957) and is suggestive that these patients had coronary artery disease.

T wave changes. Significant changes in the T wave were observed in 24 instances after induction with thiopentone, and in 2 of these cases (nos. 43, 45) there were the associated ST segment changes previously discussed.

The incidence of T wave changes varied in the different leads as illustrated in table XIII.

TABLE XIII

Lead	Total no. of cases	No. of cases seen in 1 lead only
I	8	1
II	12	3
V1	3	0
V3	15	8
V5	5	1

In 4 cases (nos. 2, 7, 32, 45) the T wave changes in the chest leads were associated with a change of sign.

In lead I seven of the T wave changes were depressions but in one (case no. 7) the T wave was raised.

In lead II the T wave was depressed in 10 instances and raised in 2 (case nos. 7, 45).

No significant changes were observed in lead III. In lead V1 the T wave was raised in 2 instances (case nos. 2, 37) and depressed in 1 (case no. 7). Cases nos. 2 and 7 were associated with a change of sign.

In lead V3 the T wave was depressed in 9 cases and raised in 6. Three cases (nos. 2, 32, 45) were associated with a change of sign.

In lead V5 the 5 cases (nos. 10, 18, 23, 34, 37) showing T wave changes were all depressions and were not associated with a change of sign.

It will be seen that the general effect of thiopentone induction is to produce T wave depressions in leads I, II and V5, no change in lead III and a variable response in chest leads V1 and V3.

In the one case (no. 37) where significant changes were seen in 5 of the leads, the T wave depressions in leads I, II and V5 were associated with T wave elevations in leads V1 and V3.

The T wave changes could not be related to the sex, age or weight of the patients, to the dose of thiopentone used, or to those who had received chlorpromazine as part of their premedication.

Three of the 4 hypertensive cases in the series (nos. 28, 34, 43) and both cases with cardiomegaly (nos. 28, 45) showed T wave changes.

The T wave changes were associated with a rise in pulse rate in 15 cases and in 1 case (no. 37) the pulse rate was doubled, in 5 cases there was a decrease in pulse rate, and in 4 cases there was no change.

All the cases showing T wave changes except one (case no. 10) were associated with a fall in systolic pressure, the average fall being 29.6 mm Hg, and it is possible that the T wave changes were due to a reduction in coronary flow.

It is known that marked respiratory depression following thiopentone induction can result in hypercarbia and a rapid increase in the plasma potassium level with resultant T wave changes, but it is felt that no such marked depression of respiration followed induction with thiopentone in this series.

Arrhythmias. These were observed in 3 cases (nos. 1, 5, 40) after thiopentone induction and consisted of ventricular extrasystoles, although extrasystoles—1 supraventricular (case no. 11) and 3 ventricular (case nos. 24, 35, 38)—which were present prior to induction disappeared after induction with thiopentone.

Although auricular arrhythmias are reputed to be uninfluenced by thiopentone (Johnstone, 1951), case no. 11 proved an exception to this rule. The arrhythmias which disappeared after thiopentone (case nos. 11, 24, 35, 38) were all associated with a fall in systolic pressure. Case no. 35 was associated with an increase in pulse rate, case no. 11 with a decrease in pulse rate, and case nos. 24 and 38 with no change. In the conscious subject the disappearance of ventricular extrasystoles, whether as a result of effort, amyl

nitrite or atropine, is usually associated with an increase in pulse rate.

The arrhythmias which appeared after thiopentone induction (case nos. 1, 5, 40) were also associated with a fall in systolic pressure. Case nos. 1 and 5 were associated with no change in the pulse rate and case no. 40 with a decrease.

No cases showing arrhythmia had thyroid disease although 6 cases in the series (nos. 18 to 23) had thyroid disease but revealed no arrhythmia.

Other investigators (Betlach, 1938; Carraway, 1939; Johnstone, 1951; Harrison, 1953; Stephen et al., 1953) have reported ventricular ectopic beats after thiopentone induction, and these have been found in the absence of CO_2 accumulation, so it is possible that they are due to the effect of the drug itself on the heart or to noradrenaline release in response to the fall in b.p.

No correlation could be found between the arrhythmias and the sex, age or weight of the patients, with the dosage of thiopentone used, with chlorpromazine premedication, or with those showing ST and T wave changes, suggesting that ischaemia is not the precipitating factor.

SUMMARY

The effect of thiopentone induction on the electrocardiogram has been studied in a series of 52 unselected cases.

The dose of thiopentone used was in general smaller in those cases receiving chlorpromazine as part of their premedication.

The general effect of thiopentone on the blood pressure was to cause it to fall, the average fall in systolic pressure being 26 mm Hg.

The general effect on the pulse rate was to increase the slower rates and to decrease the faster ones.

The effect on the P wave was to increase its height. This was significantly increased in 6 cases. The changes were seen in leads II and III. No case of nodal rhythm occurred. The P wave changes were associated with an increase in pulse rate.

There were no significant changes in the PQ or QRS intervals, but the QTc interval changed in 18 cases, in 16 cases it was prolonged and in 2 cases it was shortened.

In 12 cases there was a significant change in the electric axis and in 6 cases there was a significant change in the QRS electric vector.

One case showed a significant change in the ST segment only and 2 cases showed significant changes in both the ST segments and T waves. Twenty-two cases showed significant changes in the T wave only so that in all 24 cases showed significant T wave changes.

Arrhythmias appeared in 7 cases. In 4 cases the ectopic beats disappeared after induction with thiopentone, and in 3 cases ectopic beats followed induction.

The significance of the various changes is discussed.

ACKNOWLEDGMENTS

Our thanks are due to Mrs. A. Williams, A.S.C.T., and Miss M. C. Hirst, A.S.C.T., for recording the electrocardiograms; to our surgical colleagues Mr. J. R. Blackburne, F.R.C.S., Mr. R. R. Simpson, F.R.C.S., and Mr. J. N. Young, F.R.C.S., for their co-operation, and to Mr. D. Patchett and Miss N. M. Collingwood for technical and secretarial help respectively.

REFERENCES

Beck, C. S. (1957). Symposium on coronary artery disease. Blood supply to ischaemic myocardium distal to the occlusion of a coronary artery. *Dis. Chest*, **31**, 243.

Betlach, C. J. (1938). The effects of pentothal sodium on the electrocardiogram of patients with essential hypertension. *Proc. Mayo Clin.*, **13**, 189.

Burch, G. E., and Winsor, T. W. (1949). *A Primer of Electrocardiography*, 2nd ed. Henry Kimpton: London.

Carraway, B. M. (1939). Pentothal sodium with nasal oxygen: a report of 3810 consecutive cases. *Curr. Res. Anesth.*, **18**, 259.

Hale, D. E. (1954). *Pentothal Sodium Anesthesiology*, 1st ed. Philadelphia: F. A. Davis Co.

Harrison, G. (1953). Arrhythmia during kemithal anaesthesia. *Brit. J. Anaesth.*, **25**, 204.

Johnstone, M. (1951). Pulse irregularities during thiopentone anaesthesia. *Anaesthesia*, **6**, 138.

Rollason, W. N., and Cumming, A. R. R. (1956). The electrocardiogram in hypotensive anaesthesia. *Anaesthesia*, **11**, 319.

—— Hough, J. M. (1957). Electrocardiographic studies during endotracheal intubation and inflation of the cuff. *Brit. J. Anaesth.*, **29**, 363.

Ruth, H. S., Tovell, R. M., Milligan, A. D., and Charleroy, D. K. (1939). Pentothal sodium: is its growing popularity justified? *J. Amer. med. Ass.*, **113**, 1864.

Stephen, C. R., Martin, R., and Nowill, W. K. (1953). Cardiovascular reactions to surital, pentothal, or evipal combined with muscle relaxants for rapid anaesthesia induction. *Curr. Res. Anesth.*, **32**, 361.

Volpitto, P. P., and Marangoni, B. A. (1938). Electrocardiographic studies during anaesthesia with intravenous barbiturates. *J. Lab. clin. Med.*, **23**, 575.

Wood, P. (1956). *Diseases of the Heart and Circulation*. London: Eyre and Spottiswoode.

Brit. J. Anaesth. (1958), **30**, 63

MEDIASTINAL EMPHYSEMA

BY

JUDITH N. NICHOLAS

St. James' Hospital, Balham, London SW2

INTRODUCTION

THE occurrence of pneumomediastinum is quite common and its effects are usually benign, but may be serious. As awareness of the pathogenesis of interstitial and mediastinal emphysema is as important as clinical recognition for its prompt effective management, the subject has been reviewed with particular reference to the mechanism of production of pulmonary oedema, of which there are few clear-cut clinical reports to be found (Case I).

AETIOLOGY

(Hamman, 1945; Wallace, 1946)

(1) *Increase of Intrapulmonary Pressure resulting from Forceful Respiratory Efforts against Pressure.*

(*a*) Occlusion, partial or complete, of trachea or bronchi, particularly with cough.

(i) Complication of foreign body in a bronchus (Forbes, 1926; Hammond, 1944; Macklin and Fisher, 1940).

(ii) Asthma and chronic bronchitis.

(iii) Whooping cough.

(iv) Influenza, (v) Pneumonia (Berkeley and Coffen, 1919; Torrey and Grosh, 1919).

(vi) Diphtheria, (vii) Laryngotracheobronchitis (Graebner, 1939; Johnson, 1944; McMath, 1956).

(*b*) Positive pressure.

(i) Closed methods of anaesthesia with or without an endotracheal tube (Marcotte et al., 1940). Intermittent positive pressure respiration possibly.

(ii) Inhalation therapy, including neonatal resuscitation (Collins, 1948; Emery, 1956; Salmon et al., 1947).

(iii) Lung blast and caisson workers (Macklin and Macklin, 1942).

(*c*) Straining with the glottis closed.

(i) Laryngospasm, partial or complete.

(ii) During parturition.

(iii) Straining at stool.

(iv) Heavy lifting.

(*d*) Pre- and postoperative complication of tracheotomies, for laryngeal obstruction (Forbes and Salmon, 1946; Forbes et al., 1947; Graebner, 1939; Johnson, 1944; McMath, 1956; Michels, 1939; Neffson, 1943; Work, 1943).

(2) *Trauma* (Gaudrault and Chalmers, 1941; Jessup, 1931; Macklin and Macklin, 1942; Wallace, 1946).

(*a*) Injury to the chest, with or without fracture of the ribs (i.e. closed).

(*b*) Operations on the chest, particularly induction of pneumothorax.

(*c*) Complication of penetrating wounds of trachea and bronchi.

(3) *Spontaneous Rupture of the Alveoli* (Fisher, 1941; Hamman, 1937, 1939, 1945; Hayes, 1941; Morey and Sosman, 1939).

The precipitating conditions in the presence of the above causes fall into three categories (Macklin and Macklin 1944):

(*a*) There is first an atelectasis of some part of the lung, followed by hyperinflation in adjoining regions of the same or in the opposite lung.

(*b*) There is a general overinflation with or without increased intra-alveolar pressure.

(*c*) There is a decreased bloodflow through the pulmonary blood vessels, usually with increased intra-alveolar pressure or with hyperinflation.

EXPERIMENTAL STUDIES

The mechanism of production and spread of interstitial emphysema of the lungs has been described by Macklin (1937, 1939; Macklin and

Macklin, 1944) in his experiments on cats as follows:

Interstitial emphysema of the lungs may be produced by blowing air into a local region of the lung causing alveolar hyperinflation. Only marginal alveoli whose bases rest against bronchi, bronchioles, blood vessels, connective tissue septa and pleura are involved in the process. Those whose bases rest against adjoining alveoli are not affected. Increase in the size of marginal alveoli by hyperinflation and raised intra-alveolar pressure or decrease in size of adjacent pulmonary blood vessels leads to rupture of alveolar bases, so causing a decreasing pressure gradient and the progressive passage of air from the alveoli to the pulmonary perivascular connective tissue sheaths. Air continues to pass from the former to the latter, then follows the path of least resistance as it travels along the sheaths in blebs of increasing size towards the hilum of the lung. In frozen sections this air surrounding the sheaths has been demonstrated; the vessels are encroached upon and may even be collapsed by air bubbles. Further leakage causes the accumulating air to break through into the mediastinum, distending its tissues with large blebs.

Over fifteen years ago, at a time when the insufflation technique of anaesthesia was popular, reports of unexplained deaths during and following endotracheal anaesthesia stimulated the study of differential intrabronchial pressure in the production of mediastinal emphysema in cats and dogs. The intrabronchial pressure was regulated by the amount of resistance to the escape of gas from the lungs during endotracheal anaesthesia with a continuous air-ether stream. It was found that a pressure of 15 mm Hg in cats and 24 mm Hg in dogs was sufficient to produce mediastinal and sometimes subcutaneous emphysema and, with markedly increased intrabronchial pressure, right cardiac failure. It was noted that emphysema, once produced, did not require so great a pressure for its further development. These authors stated that too often endotracheal anaesthesia was being employed with increased intrabronchial pressure when a less hazardous technique would suffice, and emphasized that intrabronchial pressure depends chiefly upon the patency and resistance of the outlet rather than the pressure at which the gases are delivered to the lungs.

PATHS BY WHICH AIR REACHES THE MEDIASTINUM

(1) *From interstitial tissues of the lung.* This is the common route of entry and others are rare and of little practical importance in comparison.

(2) *Along deep fascial planes of the neck* (Forbes et al, 1947; Work, 1943). This *may* be the path followed during tracheotomy, thyroidectomy, tonsillectomy or puncture and irrigation of the maxillary antrum and in deep wounds of the neck, the pharynx and buccal cavity, but in the first two instances, anaesthesia and obstruction play an important part in producing interstitial pulmonary emphysema as above.

(3) *Through perforation of trachea, bronchus or oesophagus into mediastinum.* Disease or trauma occasionally causes rupture of the lower trachea or a main bronchus, and sometimes mediastinal emphysema is seen after lobectomy or pneumonectomy. If air enters from the oesophagus, symptoms of infection obscure those of air in the mediastinum (Furstenburg and Yglesias, 1937).

(4) *From the retroperitoneal space.* If air accumulates in the retroperitoneum it may travel along the aorta or the oesophagus into the mediastinum. Perirenal insufflation in radiography of kidneys and adrenal glands has often been the cause of mediastinal emphysema and it may also follow perforation of stomach or intestine.

ESCAPE OF AIR FROM THE MEDIASTINUM

When sufficient pressure develops, air always escapes from the mediastinum. Sometimes, however, it escapes when there is little increase in pressure and at other times is retained when there is evidence of great increase in pressure. Apparently the location of air within the mediastinum is an important factor, escape being easy from certain situations (e.g. it travels with ease along perivascular connective tissue) and difficult from others. There are five avenues of escape:

(1) Into the root of the neck.

(2) Into the retroperitoneum along the aorta or oesophagus.

(3) Forward between parietal pleura and pericardium to appear as blebs of air overlying the heart.

(4) Laterally into the interstitial tissue of the opposite lung or unbloated parts of the same lung, splinting them.

(5) Rupture through the mediastinal pleura into the ipsilateral or contralateral pleural cavity to produce a pneumothorax more commonly on the left.

EFFECTS OF PULMONARY INTERSTITIAL AND MEDIASTINAL EMPHYSEMA

These are dependent upon the amount of air and an available route of escape. When there is extensive subcutaneous emphysema the symptoms of mediastinal pressure are apt to be slight, whereas when these symptoms are grave there is usually little subcutaneous air. If the air cannot escape from the mediastinum and the pressure rises too high, the condition becomes malignant.

(1) *Pain.*

Pain is produced when air has reached the mediastinum in sufficient quantity to distend its tissues (Scott, 1946). It is anginal in character, often very severe and is usually located beneath the sternum (Hamman, 1939), and may be accompanied by a sensation of pressure or of expansion beneath the sternum; sometimes it radiates to the infrascapular region and at others into the neck, shoulders or more rarely to the angle of the jaw or arms.

(2) *Respiratory Effects.*

The splinting action of the air in the connective tissue of the lung interferes with respiration, causing airlock, preventing the escape of air in expiration and giving rise to dyspnoea and cyanosis.

The effects of air lock (pulmonary interstitial emphysema) and airblock (mediastinal emphysema) have been well described in reports of patients with influenzal bronchopneumonia in the 1918-19 pandemic (Berkeley and Coffen, 1919; Torrey and Grosh, 1919). Some patients, many of whom died, showed intense dyspnoea and cyanosis with little cardiac disturbance, great air hunger and erection of the chest fixed in a state of hyperinspiration with only tidal air movement. As this state developed, the whole excursion of the chest was noted to diminish until there was inspiratory retraction, more on the affected side at the onset, then on both sides, until the accessory muscles of respiration were the only ones lifting the upper chest. There was marked sternal tympany and stasis of veins entering the chest, with ashen lividity, increasingly profound cyanosis and accompanying mental depression in the end stages. Some of these patients developed subcutaneous emphysema, following which the dyspnoea and cyanosis were relieved and recovery ensued. Findings of mediastinal and subcutaneous emphysema were confirmed at postmortem examination, but there was no evidence of tears in the visceral pleura to account for the pneumothorax which had occurred in some.

Macklin's experiments (1937, 1939; Macklin and Macklin, 1944) showed that vigorous positive pressure inflation or insufflation of the lungs was followed by pneumothorax and collapse after the break through of air from mediastinum to pleura and was often accompanied by spreading subcutaneous emphysema. In most instances the air reached the pleural cavity by way of a tear in the delicate wall of the mediastinum, often demonstrable in the posterior mediastinal wall at varying levels, most commonly just below the hilar region. He immersed the lungs in water and compressed them, but was never able to demonstrate visceral pleural tears, nor could he force air from the pleura into the mediastinum. Barrie (1940) found experimentally that post mortem even moderate pressure inflation of the mediastinum produced pneumothorax, the air usually escaping through a rupture of the pleura just above the hilum of the left lung. There is also a region in the posterior mediastinum between oesophagus and aorta in which two layers of mediastinal pleura are in apposition, and rupture at this point may put the two pleural cavities into communication, producing bilateral pneumothorax. Hamman (1937, 1939, 1945) considers that this might be the mechanism of production of spontaneous pneumothorax and account for the build-up of tension.

Following injury to the chest there is often great shortness of breath and cyanosis. Pneumothorax may be found and pressure in the pleural cavity may be high; in many cases there is also

subcutaneous emphysema of the neck, demonstrating the presence of air in the mediastinum. It is probable that the injury may not have ruptured the visceral pleura, but caused alveolar rupture with formation of interstitial emphysema first of all, the air reaching the pleura by way of the mediastinum. This would also account for the occurrence of "contralateral" pneumothorax difficult to explain by assuming the existence of any other pathway.

(3) *Circulatory Effects.*

In interstitial emphysema, the air sheathing the pulmonary vessels interferes with circulation in the lung, promoting cyanosis and dyspnoea. A rise in mediastinal pressure, particularly if sudden, may have alarming and even fatal results. Clinical observation (Aisner and Franco, 1949; Salmon et al., 1947; Wallace, 1946) demonstrates clearly the circulatory embarrassment superimposed on the existing respiratory difficulties. Air under pressure in the mediastinum surrounds and obstructs the vessels, first the pulmonary veins and then the arteries and venae cavae. More blood is forced into the lung than can escape and the pulmonary capillary pressure gradually rises. Transudation of fluid from them occurs and acute pulmonary oedema results (Macintosh and Mushin, 1940). Experimentally (Ballon and Francis, 1929) there are rapid changes when mediastinal pressure reaches 0 mm H_2O pressure; there is a fall in blood pressure, tachycardia, pneumothorax and death from pulmonary oedema at 35 mm H_2O pressure.

CLINICAL FEATURES OF MEDIASTINAL EMPHYSEMA
(Hamman, 1939, 1945)

(1) It may be spontaneous, occurring without the least effort when the patient is quietly standing, sitting or lying down.

(2) When subcutaneous emphysema appears in the neck under circumstances in which there is no other cause for it, the diagnosis is at once certain. It is conclusive and sometimes the *only* evidence for its existence.

(3) Pain and substernal pressure; variety of radiation; pain on swallowing from the pressure of air around the oesophagus when the air extends retroperitoneally.

(4) No constitutional symptoms or evidence of shock accompany the pain unless circulatory failure supervenes. Temperature, respiratory rate, blood pressure and leucocyte count are little altered as a rule.

(5) There is diminution in or obliteration of cardiac dullness and replacement by a hyperresonant percussion note.

(6) Peculiar and pathognomonic sounds are heard over the praecordium, synchronous usually with cardiac systole (Hamman's sign) but sometimes also with diastole. The sounds are variously described as crepitant, crunching, crackling, bubbling or clicking. Sometimes they are heard only with the patient in certain positions, e.g. lying on the left side or sitting, and occasionally may be heard by the patient or as an extra-auscultatory sound by the examiner.

(7) Evidence of increased mediastinal pressure: dyspnoea, cyanosis, engorged veins, rapid pulse, fall in blood pressure, pulmonary oedema, circulatory failure.

(8) Pneumothorax is often found; it is usually small, is more common on the left, and may not be suspected before radiological examination. Tension pneumothorax may develop. In the presence of interstitial emphysema the size of a pneumothorax may have no relationship to its pressure. The lung, splinted by interstitial air may be prevented from more than a moderate degree of collapse and the pneumothorax may have a very high tension, the evidence for which is shift of the mediastinum and depression of the diaphragm.

(9) Radiography of the chest is valuable in establishing diagnosis. In instances where the characteristic sound over the heart is absent, radiological evidence of air in the mediastinum may be decisive. It appears in the postero-anterior film as a sharp, distinct line running parallel to the left and sometimes also to the right border of the heart and is interpreted to be the outer wall of the mediastinum (Macklin, 1937; Morey and Sosman, 1939). The most convincing picture is seen in the lateral film when the tissues between the heart and anterior chest wall are infiltrated with air. Extensive mediastinal emphysema may, however, be present, although a lateral film does not always show a substernal collection of air (Salmon et al., 1947). Pneumothorax alone, particularly in babies, if under tension may cause an apparent collection of substernal air, probably

because of herniation of the anterior mediastinum (Collins, 1948; Salmon et al., 1947).

(10) Electrocardiographic changes are absent in most cases (Aisner and Franco, 1949; Klein, 1947). About 25 per cent of patients show abnormalities which are not specific for this condition. Significant changes are those in the T waves, including inversion in all conventional leads, deviation of the S-T segment from the iso-electric level, absent or greatly reduced initial deflection in standard lead IV, decreased voltage and shifts in the electrical axis (Littman, 1946). Pneumothorax per se may produce similar changes. Any alterations are usually transient, reversion to normal taking place in seven to fourteen days.

DIFFERENTIAL DIAGNOSIS

(Aisner and Franco, 1949; Hamman, 1934; Hayes, 1941)

A differential diagnosis must be made between mediastinal emphysema and the following:

Coronary occlusion.
Angina pectoris.
Pericarditis.
Spontaneous pneumothorax (may accompany).
Pulmonary embolism.
Dissecting or ruptured aortic aneurysm.
Ruptured oesophagus.
Mediastinitis.

TREATMENT

(Collins, 1948; Hammond, 1944)

Conservative.

(1) Bedrest.

(2) Control of cough or strain.

(3) Management of pulmonary oedema when it occurs.

(4) (*a*) Aspiration of air from a large pneumothorax if it is embarrassing respiration.

(*b*) Aspiration of air from the anterior mediastinum if subcutaneous emphysema is spreading or if the circulation is seriously embarrassed. This should be tried before any operative measures. A needle or cannula may be inserted parallel to the inferior surface of the sternum through the third left or fourth right intercostal space (Collins, 1948) or the second or third intercostal space just lateral to the sternum (Forbes et al., 1947).

Radical.

(1) Tension pneumothorax demands aspiration of air under tension and closed thoracotomy with an intercostal catheter connected to an underwater seal.

(2) (*a*) Multiple subcutaneous aspiration of air through needles.

(*b*) Multiple incisions into the subcutaneous tissues where the air has accumulated.

(3) A continuing leak from lung to mediastinum has sometimes been successfully treated by the induction of artificial pneumothorax to collapse the lung and seal the leak (the value of this is doubtful).

(4) Cervical mediastinotomy may be necessary if aspiration of the mediastinum is successful but not sufficient. A transverse incision above the suprasternal notch may be made or a longitudinal one from the thyroid notch to the sternum and blunt dissection carried down to the trachea and laterally to the carotid vascular sheaths (Hammond, 1944) and deep to the sternum.

(5) Splitting of the sternum has on occasion been necessary (Hamman, 1945).

PROGNOSIS

Most cases of mediastinal and subcutaneous emphysema and spontaneous pneumothorax are benign and recover with rest alone. The more serious and prolonged complications require a guarded prognosis; that of tension pneumothorax depends on the success of early treatment and absence of further complications; that of acute heart failure varies with the promptness of its relief and the presence of any residual signs; while that of spreading subcutaneous emphysema depends on the rapidity with which it is relieved.

CASE REPORTS

Over the past few years there have been several reports of cases of subcutaneous and mediastinal emphysema and of pneumothorax occurring during induction of anaesthesia or during operation (Dundee, 1955; Fairley, 1955; Rollason, 1951, 1955; Spence, 1955). In the following series there are six cases seen over a period of three years, and of these, two had very grave symptoms and one died.

CASE 1

A girl aged 30 was admitted for excision of a solitary thyroid nodule. She had had a tuberculous lesion at the apex of the right lower lobe treated seven years earlier by repeated artificial pneumothoraces. Thyrotoxic signs were absent and the heart and lungs were clinically normal. A pre-operative chest radiograph showed a healed tuberculous lesion (fig. 1).*

Anaesthesia was induced with intravenous thiopentone and gallamine triethiodide and maintained with nitrous oxide and oxygen delivered by face mask, and respiration was assisted throughout.

Operation was of one hour's duration. Quite early, slight cyanosis of the fingertips was noted, but not of the blood in the wound. There was at intervals some mild degree of respiratory obstruction, as may often occur during thyroidectomy performed without an endotracheal tube. Towards the end of the operation definite cyanosis was noted which did not respond to pure oxygen and assistance to respiration. The airway was clear and there was no obvious cause for the cyanosis. Operation was completed rapidly, and intravenous atropine and neostigmine were given.

On examination the patient was conscious but dyspnoeic and stated that she felt she could not take a deep breath; she was ashen and sweating and the nailbeds were deeply cyanosed, while there was frothy aspiration from the mouth and pharynx. The pulse was faint and regular, its rate 120 per minute; the apex beat was inside the nipple line in the fifth left intercostal space and the blood pressure was 70 mm Hg systolic. The tidal volume was small and there was inspiratory retraction in the left upper intercostal spaces, and on auscultation there were moist râles throughout the lung fields. There was no stridor. The voice was weak, but phonation was unimpaired. The diagnosis of acute pulmonary oedema was made, and oxygen was given by facemask with assistance to respiration with the patient supported in the sitting posture. Aminophylline 240 mg and papaveretum 10 mg ($\frac{1}{6}$ grain) were given intravenously.

One hour after operation, when seen by the consultant physician, the systolic blood pressure had fallen to 40 mm Hg and the other signs were unchanged. He recommended the commencement of digitalization with 0.5 mg of intravenous digoxin, further sedation and the administration of 10 mg of methylamphetamine, in view of the extreme degree of circulatory failure.

Over the next seven hours the patient complained of pain on swallowing and of substernal pressure. Improvement was gradual, the blood pressure rising slowly to 85/60 mm Hg and the pulse rate remaining at 140 per minute. The treatment was continuous intranasal oxygen, four doses of 10 mg of methylamphetamine and two doses of papaveretum 20 mg ($\frac{1}{3}$ grain) intravenously.

On re-examination there was pallor, slight cyanosis, pin-point pupils and no sweating. The pulse rate was 136 per minute, the blood pressure 80/60 mm Hg, and the respiratory rate 18 per minute. The apex beat was now found outside the nipple line five inches from the midline in the fifth intercostal space on the left. The heart sounds were distant, there was tictac rhythm, and a crunching sound synchronous with systole could be heard in all areas. There was no visible or palpable subcutaneous emphysema. Respiratory movement was now easier, equal on right and left, and intercostal retraction had disappeared. The percussion note was resonant and the breath sounds vesicular and accompanied by a few crepitations in the right midaxillary line. The pulmonary oedema appeared to have subsided but there was evidently cardiac enlargement. At this time, eight hours after operation, the patient had improved sufficiently for her bed to be moved from the recovery room to the ward, and a 0.5 ml test dose of Neptal was given. This was followed later by 1.5 ml of Neptal, and the intravenous papaveretum 20 mg ($\frac{1}{3}$ grain) was repeated. Intranasal oxygen continued. The prognosis improved further with the passage of 6 ounces (170 ml) of urine ten hours after operation and another 14 ounces (400 ml) at thirteen hours. Overnight, oxygen was continued and the pulse rate varied from 128 to 140 per minute, and the blood pressure from 70/50 to 110/70 mm Hg.

On examination twenty-four hours after operation, subcutaneous emphysema was noted in the neck for the first time, the systolic crunch was still present, and the patient was greatly improved clinically, still pale but no longer cyanosed. Oxygen was continued for the next twenty-four hours, digitalis administration continued, and three doses of intravenous papaveretum

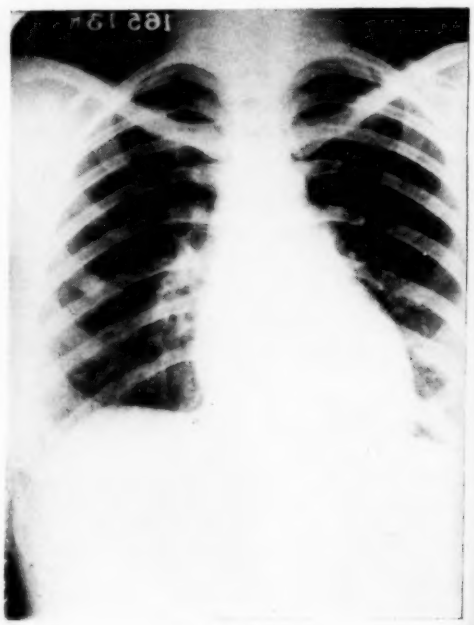

FIG. 1

Pre-operative chest film, 5.4.54. There is an old tuberculous lesion at the apex of the right lower lobe.

* Figures 1 to 6 are photographs of the relevant chest radiographs. Figures 7 to 10 are photographs of electrocardiographic tracings.

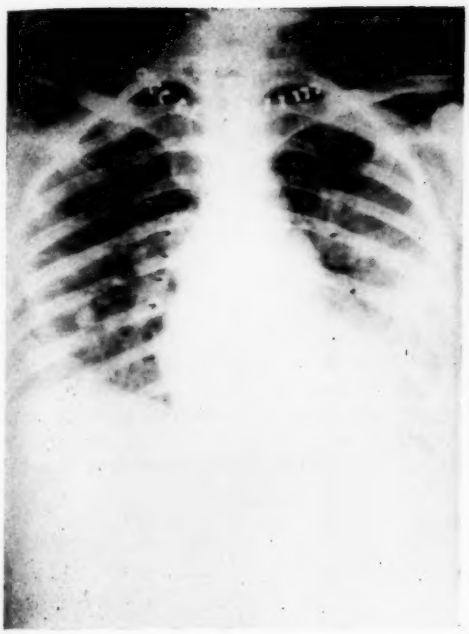

FIG. 2

FIG. 3

Second postoperative day, 9.4.54. Postero-anterior and lateral films of the chest. There is subcutaneous emphysema, cardiac enlargement and mediastinal emphysema. A streak of air is seen across the heart in the lateral film.

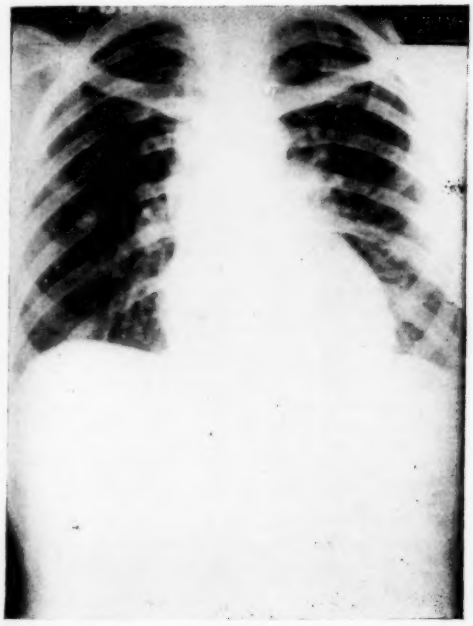

FIG. 4

FIG. 5

Fifth postoperative day; 12.4.54. Postero-anterior and lateral films of the chest. Cardiac enlargement; the mediastinal emphysema is less.

FIG. 6
Eighteenth postoperative day, 25.4.54. Postero-anterior film. The heart is still enlarged.

20 mg (⅓ grain) and one of methylamphetamine 10 mg given. The pulse rate dropped to 120 per minute and the blood pressure remained steady at or above 80/60 mm Hg. Postero-anterior and lateral radiographs of the chest were taken and showed cardiac enlargement and mediastinal and subcutaneous emphysema (figs. 2 and 3). The electrocardiogram showed a Q wave in lead I and inverted T waves, with left ventricular preponderance (figs. 7 and 8).

From the third postoperative day recovery progressed slowly. On the fifth day, the mediastinal emphysema was less, but the cardiac enlargement still gross (figs. 4 and 5). By the eighteenth day, the air had disappeared in the chest films but the heart was still enlarged (fig. 6). Because of the acute nature of the cardiac failure and its accompanying radiographic and electrcardiographic signs, bedrest was continued for six weeks and progress was assessed both by clinical improvement and return of heart size and electrocardiographic tracing to normal. On discharge there was still slight cardiac enlargement and persistent inversion of T waves in leads I and II (figs. 9 and 10). At follow-up after eleven weeks, both heart size and tracing had returned to normal and work was resumed after thirteen weeks.

Discussion.

In this case the condition arose during closed circuit anaesthesia with intermittent positive pressure inflation of the lungs during operation; there may have been raised intrabronchial pressure caused by a mild degree of unappreciated obstruction preventing the escape of gases in the lungs. At the close of the operation there was acute pulmonary oedema with an extreme degree of circulatory failure; symptomatic treatment of this was prompt, but what probably saved the patient's life was that the cessation of positive pressure inflation and resumption of spontaneous breathing at the end of operation checked the continued passage of air from alveoli to mediastinum, as a pressure gradient no longer existed between them.

It was suggested that the air had entered from the neck during dissection and a post-operative follow-up postero-anterior chest film within twenty-four hours was taken in the next twenty consecutive routine thyroidectomies. None showed air in the mediastinum and only one showed any in the neighbourhood of the wound.

Barrie (1940) reported three fatal cases all noted to be cyanosed during thyroidectomy. All developed bilateral pneumothorax and collapse with subcutaneous emphysema and air in the anterior and superior mediastinum demonstrated post mortem. There were similar findings in Work's nonfatal case (Work, 1943).

CASE II

A year later, another case of mediastinal emphysema occurring during thyroidectomy was seen. The patient was a healthy woman of 40 with a nodular goitre.

Induction of anaesthesia was smooth and maintenance was with nitrous oxide and oxygen, delivered through a no. 9 cuffed endotracheal tube, and intravenous pethidine supplementation as necessary. Respiration was spontaneous, unassisted and entirely unobstructed until, during ligation of the superior thyroid artery and mobilization of the upper pole of the right lobe, which was difficult, the surgical manipulation was responsible for complete obstruction of the endotracheal tube for perhaps 15 to 30 seconds. This was recognized and relieved immediately. At the end of operation there was tachycardia, and subcutaneous emphysema was observed in the neck. Further signs of mediastinal emphysema were sought and found: a crunching sound synchronous with systole, and the presence of mediastinal air in a chest film taken 4 hours after operation.

Discussion.

It was felt that the air did not enter from the neck, but from alveoli ruptured during the temporary overinflation produced by the period of

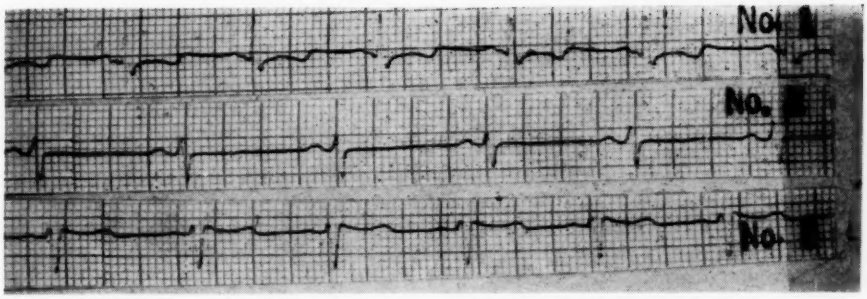

FIG. 7

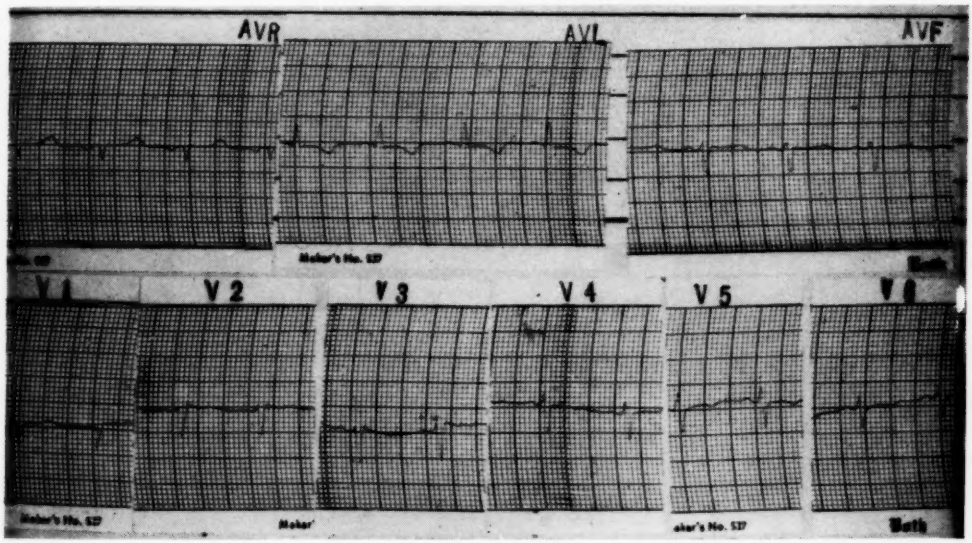

FIG. 8

Case I. Second postoperative day, 9.4.54. Lead I shows a Q wave and inversion of T waves. There is left ventricular preponderance.

complete respiratory obstruction. The condition was not further aggravated as no positive pressure was used in respiration and it pursued an entirely benign course, subsiding rapidly within a few days of operation.

CASE III

A male patient, aged 66, presented with a history of dysphagia for twenty years, increasing over the past three years, and a swelling in the left side of the neck, which could be emptied by his bending over. The heart and lungs were clinically normal and radiography showed the lung fields to be emphysematous, while a barium swallow demonstrated a large pharyngeal diverticulum extending into the superior mediastinum.

Operation for excision of the pouch was undertaken. Induction was performed with thiopentone and suxamethonium chloride, and the lungs were inflated a few times with pure oxygen delivered via a face-mask whilst the patient was apnoeic. Anaesthesia was maintained with nitrous oxide and oxygen given via a no. 10 cuffed endotracheal tube and supplemented with fractional doses of pethidine. Respiration was

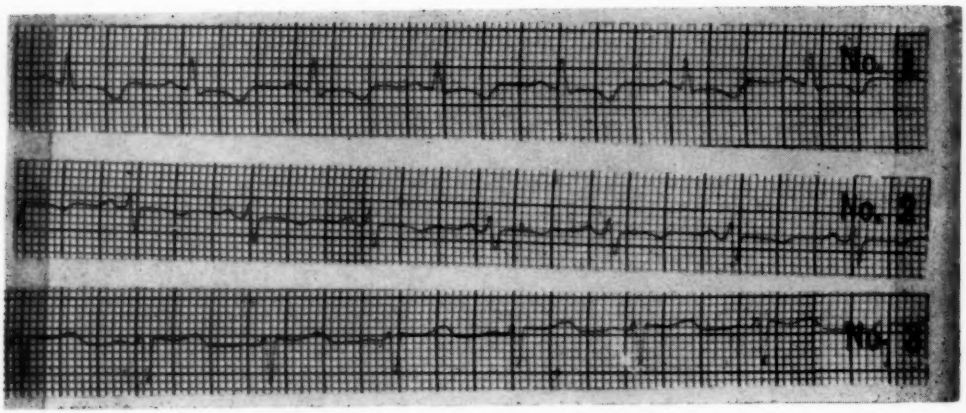

FIG. 9

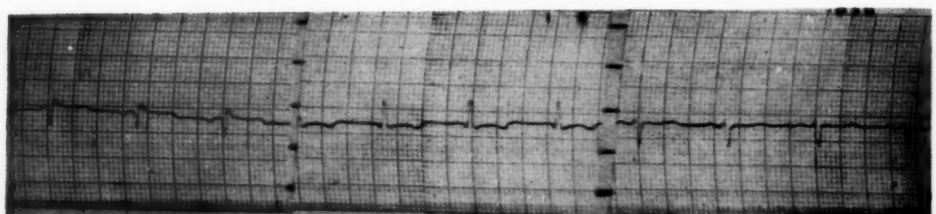

FIG. 10

Case I. Six weeks after operation. Lead I still shows inversion of T waves and there is left ventricular preponderance.

spontaneous and unassisted. Towards the end of the two-hour operation, air was noted to be bubbling in and out of the wound with the respiratory movements. The wound was closed with a corrugated rubber drain extending into the superior mediastinum.

On return from the theatre the patient was unconscious and cyanosed without respiratory obstruction or depression of depth or rate. Cyanosis showed little response to intranasal oxygen. There was subcutaneous emphysema in the right supraclavicular fossa on the side opposite that of the pouch and operation. The only abnormal sign in the chest was the presence of expiratory wheezing rhonchi over the right apex.

A provisional diagnosis of mediastinal emphysema and mild compression was made. Consciousness was not recovered until seven hours after operation. The

following morning, there was subcutaneous emphysema extending to the right axilla, the colour was pink and there was pain on swallowing. Radiography showed bilateral pneumothorax with partial collapse of both lower lobes. On the third postoperative day a systolic crunch was heard to the left of the sternum on sitting and leaning to the left. Convalescence was otherwise uneventful and the patient was discharged after three weeks, still with a small pneumothorax and fluid at the left base.

Discussion.

It was thought that the occurrence of the small bilateral pneumothoraces may have decompressed the mediastinum and caused the colour to return to pink, rather than further embarrassing respiration as might have been expected.

CASE IV

A man of 28 with a provisional diagnosis of disseminated sclerosis had vertebral arteriography carried out under general anaesthesia during the course of investigations.

Anaesthesia was with the thiopentone and suxamethonium sequence for induction, positive pressure inflation of the lungs for one minute with pure oxygen cocaine spray to the larynx, and a No. 8 Magill cuffed tube was passed with some difficulty. Maintenance was with nitrous oxide, oxygen and trichloroethylene with a semi-open circuit.

One hour from the commencement of dissection for a left vertebral arteriogram, the surgeon thought he had opened Sibson's fascia and later air could be seen and heard squeaking out from tissue planes posterolateral to the trachea, and the possibility of medistinal emphysema was entertained. Half an hour later, the pulse rate was 130 per minute, the colour pink, the trachea a little to the right of the midline while the left side of the chest appeared to be more inflated than the right and was hyperresonant and breath sounds were diminished on that side. Through the endotracheal tube an element of inspiratory and expiratory obstruction could be heard and there was inspiratory retraction in the suprasternal notch and the intercostal spaces, more marked on the right than the left. Cardiac dullness was diminished to percussion, there was sternal tympany and both heart sounds were accompanied by a crunching noise. Operation was abandoned and antero-posterior and lateral radiographic films of the chest were taken on the operating table and these confirmed the provisional diagnosis of a collection of air in the mediastinum and subcutaneous emphysema around the wound. Another film taken two hours later in the sitting posture showed a left pneumothorax.

Air under tension was taken from the left pleural cavity, but cyanosis of lips, nailbeds and tachycardia were unrelieved, and it was thought that the mediastinum should be needled; air under pressure, however, could not be withdrawn through a wide bore needle.

In the following forty-eight hours, fever and crepitant râles appeared, but respiratory distress did not increase. On the third postoperative day, the patient became comatose (as he had been twice before this admission) and died on the fifth day. Postmortem examination showed left pneumothorax and bronchopneumonia and confirmed the diagnosis of disseminated sclerosis.

Discussion.

The air may have reached the pleura in three ways. Firstly, alveoli may have been ruptured during positive pressure inflation, or more likely due to the relative respiratory obstruction and resistance caused by the use of a no. 8 tube in an adult male. Secondly, air may have reached the superior mediastinum from the wound, and so the left pleural cavity. Thirdly, it may have entered the pleural cavity directly, but if so, with the wound open above, could not have accumulated enough pressure also to rupture into the mediastinum. The resistance theory seems the most likely.

CASE V

A boy of 9 years with a history of tonsillitis and cough, treated with penicillin a week previously, was admitted for correction of strabismus.

After premedication with quinalbarbitone and atropine, anaesthesia was induced with a thiopentone, ethyl chloride, open ether sequence. During the introduction of the ether he coughed for about two minutes and this was followed by incomplete laryngospasm with some cyanosis relieved by inflation with half a dozen breaths of oxygen by bag and facemask and without difficulty, the colour returning to normal. Smooth intubation with a no. 5 plain Magill endotracheal tube was followed by nitrous oxide–oxygen–ether maintenance with a T-piece. Respiration was free from obstruction and anaesthesia smooth during the twenty-minute operation.

During recovery, when supporting the jaw, crepitus was felt under the skin and on examination there was invisible but palpable subcutaneous emphysema limited to both sides of the neck, the supraclavicular fossae and the suprasternal notch. This suggested the diagnosis of pneumomediastinum which was confirmed by further examination. The colour was pink, but the nailbeds were slightly cyanosed. The pulse rate was 120 per minute and the blood pressure 105/70 mm Hg. The apex beat was palpable in the fifth left intercostal space just outside the nipple line. Although there was sternal tympany, cardiac dullness did not seem to be decreased. At the apex, in the supine and left lateral positions, a crunching sound synchronous with systole could be heard. The respiratory rate was 22 per minute, the trachea midline, percussion note equal and resonant and air entry equal on both sides of the chest. The breath sounds were harsh vesicular in character.

Antero-posterior and lateral radiographic films of the chest showed a small amount of air in the neck along the trachea and a faint streak parallel with the upper part of the left border of the heart.

The subcutaneous emphysema did not spread and decreased over the next twenty-four hours; recovery was otherwise uneventful.

Discussion.

It is suggested that the coughing and laryngospasm in induction and possibly the inflation of the lungs through a facemask following a recent upper respiratory tract infection were sufficient to overdistend and rupture some alveoli. As the T-piece offers little resistance to respiration there can only have been a very low pressure gradient from alveoli to mediastinum during the rest of the course of anaesthesia, so that the mediastinal accumulation of air did not increase, and by escaping into the neck was decompressed so that circulatory embarrassment was slight.

A similar case of benign mediastinal emphysema with Hamman's sign was also reported to me following emergency operation for acute appendicitis in a boy of 14 who was an asthmatic and who was said to have coughed a few times during an open ether induction. This likewise subsided without incident.

CASE VI

A boy of 8 years fell out of a tree. He had a lacerated arm and complained of pain in the chest and the abdomen. On examination there was rebound tenderness over the lower abdomen and in the chest a clicking sound could be heard accompanying systole. A wet film of the chest did not show pneumothorax or mediastinal emphysema, but this was thought to be present on clinical grounds alone. It was decided that a laparotomy should be performed. This raised the question of choosing the least dangerous form of anaesthesia in the presence of ruptured alveoli, so as not to increase the amount and pressure of air in the mediastinum. To avoid the use of any positive pressure at all which would create a pressure gradient and increase the likelihood of a continuing leak, induction was with cyclopropane and oxygen, changing over to ether, nitrous oxide and oxygen smoothly. Intubation was performed with a no. 5 plain Magill endotracheal tube and the whole course of anaesthesia was smooth. Tachycardia of 130 to 140 per minute was the only sign of embarrassment to the circulation. Postoperatively, the condition subsided without incident.

CONCLUSIONS AND PREVENTION

The only basis for reducing the incidence of mediastinal emphysema and its serious complications is to recognize and avoid the predisposing causes, including postponement of operation in patients with recent respiratory tract infection, as part of the deliberate management of any patient. During operation it should be almost entirely preventable by the efforts of anaesthetist and surgeon.

To the surgeon two things are of paramount importance:

(i) He must avoid tracheal obstruction by pressure from without in dissection during thyroidectomy, vertebral arteriography, block excision of cervical glands, excision of pharyngeal pouch and oesophagoscopy. If this occurs resistance to expiration is raised sharply and dangerously high intrabronchial pressures can be produced.

(ii) He should not carry out too low a dissection for tracheotomy or separate tissue planes unnecessarily, in particular the pretracheal fascia before opening the trachea.

The anaesthetist's responsibility is even greater:

(i) A preventive contribution can be always to provide a perfectly clear airway either with a bronchoscope or endotracheal tube during emergency tracheotomy. This alone has been shown in a five-year series of cases (Forbes et al., 1947) to make a statistically significant difference to the incidence of mediastinal emphysema during and following this operation.

(ii) He must avoid scrupulously even a minor degree of respiratory obstruction and relieve it promptly when it occurs so as to avoid build-up of high pressures when there is resistance to expiration. The anaesthetic apparatus in use must also be blameless in this regard, from the use of the widest bore endotracheal tube possible to elimination of expiratory resistance by valves—for example, by the use of a T-piece. This is especially important in children.

(iii) Meticulous care must be exercised in the use of controlled respiration using intermittent positive pressure which must not be too high. The dangerous positive pressure insufflation technique responsible for many operative fatalities was abandoned after the investigations mentioned above (Marcotte et al., 1940).

(iv) Emergency taps delivering oxygen at high pressure may be very dangerous and so may the use of high rates of minute flow of gases with too little avenue of escape, i.e. too great an expiratory resistance, including too narrow an

endotracheal tube, and also the use of a "tight" bag for respiring patients.

(v) Should mediastinal emphysema occur during anaesthesia and be recognized, operation should be abandoned or rapidly terminated in order not to aggravate the symptoms arising from the accumulation of air. This applies particularly to any patient to whom the technique of assisted or controlled respiration is being applied.

(vi) If mediastinal emphysema is already present and an emergency operation for some other reason is necessary, a technique employing spontaneous respiration and no positive pressure at all should be employed in the anaesthetic management and special care paid to the avoidance of expiratory resistance.

SUMMARY

The nature, causes, effects, clinical features, diagnosis, treatment and prevention of interstitial and mediastinal emphysema are discussed together with the presentation of six cases collected over the past three years.

The prophylexis and treatment of the condition is discussed.

ACKNOWLEDGMENTS

I should like to thank Mr. F. Colahan of St. Vincent's Hospital, Melbourne; Mr. E. E. Dunlop of the Royal Melbourne Hospital, Melbourne; Mr. R. Hadley and Mr. F. Morgan of St. Vincent's Hospital, Melbourne; and Mr. I. Spiro of Hillingdon County Hospital, Middlesex, for permission to publish their cases; and Dr. J. Hayden of St. Vincent's Hospital, Melbourne; Dr. Margaret McClelland of the Royal Children's Hospital, Melbourne; and Dr. Patricia Wilson of the Royal Melbourne Hospital, Melbourne, for their encouragement, criticism and help in the preparation of this paper; and also Mr. Sullivan of St. Vincent's Hospital, Melbourne, who prepared the photographs of radiographic films and electrocardiographic tracings.

REFERENCES

Aisner, M., and Franco, J. E. (1949). Mediastinal emphysema. *New Engl. J. Med.*, **241**, 818.

Ballon, H. C., and Francis, B. R. (1929). Consequences of variations in mediastinal pressure. *Arch. Surg. (Chicago)*, **19**, 1627.

Barrie, H. J. (1940). Interstitial emphysema and pneumothorax after operation on the neck. *Lancet*, **1**, 996.

Berkeley, H. K., and Coffen, T. H. (1919). Generalised interstitial emphysema and spontaneous pneumothorax as a complication of bronchopneumonia. *J. Amer. med. Ass.*, **72**, 535.

Borrie, J. (1955). Rupture of the oesophagus. *Brit. med. J.*, **1**, 23.

Collins, V. L. (1948). Mediastinal emphysema. *Med. J. Aust.*, **1**, 614.

Dundee, J. (1955). Tension pneumothorax during the induction of anaesthesia. *Anaesthesia*, **10**, 375.

Emery, J. L. (1956). Interstitial emphysema: pneumothorax and "airblock" in the new-born. *Lancet*, **1**, 405.

Fairley, H. (1955). Tension pneumothorax complicating anaesthesia. *Anaesthesia*, **10**, 375.

Fisher, J. H. (1941). Spontaneous pulmonic interstitial and mediastinal emphysema in an infant. *Canad. med. Ass. J.*, **44**, 27.

Forbes, G. B., and Salmon, G. W. (1946). Mediastinal emphysema and pneumothorax following tracheotomy. *J. Pediat.*, **23**, 175.

—— —— Herweg, J. C. (1947). Further observations on post-tracheotomy mediastinal emphysema and pneumothorax. *J. Pediat.*, **31**, 172.

Forbes, H. H. (1926). Report of a case of emphysema of the neck and chest following inhalation of a piece of nut. *Laryngoscope (St. Louis)*, **36**, 192.

Furstenburg, A. C., and Yglesias, L. (1937). Mediastinitis: a clinical study with practical anatomic considerations of the neck and mediastinum. *Arch. Otolaryng. (Chicago)*, **25**, 539.

Gaudrault, G. L., and Chalmers, G. M. (1941). Emergency treatment of traumatic emphysema of the mediastinum. *New. Engl. J. Med.*, **224**, 940.

Graebner, H. (1939). Pneumopericardium and pneumomediastinum in cases of acute obstructive laryngitis. *Arch. Otorhinolaryng. (Chicago)*, **29**, 446.

Hamman, Louis (1934). Remarks on the diagnosis of coronary occlusion. *Ann. intern. Med.*, **8**, 417.

—— (1937). Spontaneous interstitial emphysema of the lungs. *Trans. Ass. Amer. Phycns.*, **52**, 311.

—— (1939). Spontaneous mediastinal emphysema. *Bull. Johns Hopk. Hosp.*, **64**, 1.

—— (1945). Mediastinal emphysema. *J. Amer. med. Ass.*, **128**, 1.

Hammond, A. E. (1944). Emergency cervical mediastinotomy in a case of massive mediastinal and subcutaneous emphysema secondary to removal of a foreign body from the bronchus. *Ann. Otol. (St. Louis)*, **53**, 829.

Hayes, C. W. (1941). Spontaneous mediastinal emphysema. *J. Amer. med. Ass.*, **116**, 301.

Jessup, P. M. P. (1931). Mediastinal emphysema. *Arch. Surg. (Chicago)*, **23**, 760.

Johnson, L. F. (1944). Bilateral pneumothorax in a tracheotomised infant. *Ann. Otol. (St. Louis)*, **53**, 837.

Klein, A. (1947). A spontaneous mediastinal emphysema with acute right ventricular strain. *Amer. Heart J.*, **33**, 867.

Littman, D. (1946). Electrocardiographic phenomena associated with spontaneous pneumothorax and mediastinal emphysema. *Amer. J. med. Sci.*, **212**, 682.

Macintosh, R. R., and Mushin, W. W. (1940). *Physics for the Anaesthetist*, p. 203. Oxford: Blackwell.

Macklin, C. C. (1937). Pneumothorax with massive collapse from experimental local overinflation of the lung substance. *Canad. med. Ass. J.*, **36**, 414.

Macklin, C. C. (1939). Transport of air along the sheaths of pulmonic blood vessels from alveoli to mediastinum—clinical implications. *Arch. intern. Med.*, **64**, 912.

—— Fisher, J. H. (1940). Pulmonic interstitial and mediastinal emphysema: a fatal case in a child as a result of aspiration of peanut fragments. *Amer. J. Dis. Child.*, **60**, 102.

—— Macklin, M. T. (1942). Air block and lung blast. *Lancet*, **1**, 602.

—— —— (1944). Malignant interstitial emphysema of the lungs and mediastinum as an important occult complication in many respiratory diseases and other conditions: an interpretation of the clinical literature in the light of laboratory experiment. *Medicine (Baltimore)*, **23**, 281.

Marcotte, R. J., Phillips, E. J., Adams, W. E., and Livingstone, H. (1940). Differential intrabronchial pressure and mediastinal emphysema. *J. thorac. Surg.*, **9**, 346.

McMath, W. F. T. (1956). Pneumothorax in tracheotomy for laryngotracheobronchitis. *Lancet*, **2**, 750.

Michels, M. W. (1939). Pneumothorax and mediastinal emphysema complicating tracheotomy. *Arch. Otolaryng. (Chicago)*, **29**, 842.

Morey, J. B., and Sosman, M. C. (1939). Spontaneous emphysema. *Radiology*, **32**, 19.

Neffson, A. H. (1943). Tension pneumothorax and mediastinal emphysema after tracheotomy. *Arch. Otolaryng. (Chicago)*, **37**, 23.

O'Donoghue, P. D. (1956). Mediastinal surgical emphysema due to perforated duodenal ulcer. *Lancet*, **1**, 189.

Rollason, W. N. (1951). Massive surgical emphysema occurring under general anaesthesia. *Anaesthesia*, **6**, 112.

—— (1955). Massive surgical emphysema occurring under general anaesthesia. *Anaesthesia*, **10**, 196.

Salmon, G. W., Forbes, G. B., and Davenport, H. (1947). Airblock in the newborn infant. *J. Pediat.*, **30**, 260.

Scott, A. (1946). Significance of the anginal syndrome in acute spontaneous pneumomediastinum. *Lancet*, **1**, 1327.

Spence, M. (1955). Surgical emphysema during anaesthesia. *Anaesthesia*, **10**, 50.

Torrey, R. G., and Grosh, L. C. (1919). Acute pulmonary emphysema observed during the epidemic of influenzal pneumonia at camp Hancock, Georgia. *Amer. J. med. Sci.*, **157**, 170.

Wallace, F. T. (1946). Acute pulmonary oedema associated with mediastinal emphysema. *Surgery*, **19**, 430.

Work, W. P. (1943). Mediastinal emphysema and bilateral pneumothorax complicating tracheotomy. *Arch. Otolaryng. (Chicago)*, **37**, 526.

BOOK REVIEW

Aids to Anaesthesia (4th edition). By Victor Goldman, F.F.A.R.C.S., D.A., L.R.C.P., M.R.C.S. Published by Baillière, Tindall and Cox, London. Pp. viii + 359; 92 illustrations. Price 12*s.* 6*d.*

The publication of a fourth edition of this handbook surely reflects both its popularity and the author's desire to keep the subject matter up to date with recent developments in anaesthesia.

In his introduction to this edition Dr. Goldman makes it clear that it is not intended to be an "exhaustive reference book" but an "aid" to students, house officers and postgraduates, and, with this in mind, one must agree that it fulfils its purpose admirably. In it will be found detailed descriptions of the more straightforward anaesthetic techniques, both general and regional, for coping with a variety of surgical conditions. Particularly useful to practitioners should be the excellent chapters on dental and obstetrical anaesthesia which are admirably described in a most practical and authoritative manner.

The scope and use of the current muscle-relaxant drugs in surgery and medicine is clearly illustrated and there is a chapter on the practical application of controlled hypotension. This latter chapter should perhaps be regarded by the junior anaesthetist as informative rather than as a stimulant to attempting its use. It is perhaps a little surprising that Stovaine should still be included in the armamentarium of drugs for spinal anaesthesia, although it is only fair to say that the author does not recommend its use. Similarly it is doubtful, in a book of this avowed aim, whether it is wise to persist with the description of the Etherington Wilson technique of spinal anaesthesia using light nupercaine.

These very minor criticisms, however, are in no way intended to detract from the high standard of this most useful member of the "Aids" Series.

A. G. Miller

Brit. J. Anaesth. (1958), **30**, 77

CLINICAL SIGNIFICANCE OF THE EFFECTS OF THIOPENTONE AND ADJUVANT DRUGS ON BLOOD SUGAR AND GLUCOSE TOLERANCE

BY

JOHN W. DUNDEE AND URSULA M. TODD

Department of Anaesthesia, University of Liverpool

IT has been shown, both in a previous communication (Dundee, 1956) and from the findings of other workers, that thiopentone produces no appreciable change in the blood sugar levels in man. Slight hyperglycaemia resulted when thiopentone – nitrous oxide – oxygen anaesthesia, followed morphia and atropine as premedication for operations on varicose veins. The rise in blood sugar during operation differed significantly from that produced by thiopentone alone, and it was suggested that this may have been secondary to respiratory depression produced by the morphia-thiopentone combination.

It was also shown, in the same publication, that thiopentone impairs the ability of the body to deal with an extra load of glucose. Goldsmith and Holmes (1957) have also found that a slightly greater degree of hyperglycaemia resulted from intravenous injections of dextrose given to patients under thiopentone anaesthesia than from the same dose given to the same subjects in the conscious state.

This paper is concerned with the significance of these effects of thiopentone–nitrous oxide–oxygen anaesthesia, as applied to routine anaesthetic practice. The effects of pre-operative medication and adjuvants given during thiopentone–nitrous oxide–oxygen anaesthesia are reported.

METHODS

All observations were made on fit adult patients, who had no disease other than the pathology necessitating the operation. All the anaesthetics were given personally by one of us (J.W.D.) and all the administrations were smooth throughout, with no obvious ventilatory inadequacy, hypercarbia or hypotension. Where controlled respiration was employed, the Aintree respirator was used. With the semi-open circuit, the use of gas flows of 6 litres nitrous oxide and 2 litres oxygen per minute was the standard technique. These flows were also used at the beginning of closed circuit anaesthesia, but after 10 minutes they were reduced to 2 litres of nitrous oxide and 1 litre of oxygen per minute.

Blood sugar determinations were all carried out by using the Folin-Wu technique as described by Hawk et al. (1947). Duplicate or triplicate analyses of seventeen random samples revealed an average deviation of 2.5 ± 0.40 mg/100 ml (range 0 – 5) from this mean reading. This amounts to an average error of ± 2.4 per cent with the techniques used.

RESULTS

Pre-operative medication.

Table I shows the effects of morphia (10 mg)–atropine (0.6 mg) and promethazine (25 mg)–atropine (0.6 mg) premedication on blood sugar in patients undergoing surface operations under thiopentone–nitrous oxide–oxygen anaesthesia with a semi-open circuit. These are compared with the effects of thiopentone alone. It is apparent that the use of either of these premedications, the use of nitrous oxide–oxygen or simply the performance of a surgical operation caused a slight rise in blood sugar in combination with thiopentone. Only in subjects who received morphia–atropine did the rise in blood sugar differ to a statistically significant degree. The hyperglycaemia only lasted in any case for the first hour of the anaesthesia.

The patients who were premedicated with promethazine–atropine received appreciably smaller doses of thiopentone than the morphia–

TABLE I

Average blood sugar changes during thiopentone–nitrous oxide–oxygen anaesthesia in subjects having no operation and in two groups of patients each with different pre-operative medication in whom body surface operations were performed. The average dose of thiopentone is also included.

Pre-operative medication	Nil			Morphia 10 mg; atropine 0.6 mg			Promethazine 25 mg; atropine 0.6 mg		
Operation	Nil			Surface			Surface		
Time after induction (minutes)	Number of cases	Mean deviation in blood sugar (mg/100 ml)	Mean dose of thiopentone (mg/kg)	Number of cases	Mean deviation in blood sugar (mg/100 ml)	Mean dose of thiopentone (mg/kg)	Number of cases	Mean deviation in blood sugar (mg/100 ml)	Mean dose of thiopentone (mg/kg)
20– 40	8	+0.8±4.0	10.7	8	+25.3±4.0	13.6	14	+4.0±1.6	7.9
50– 70	8	0	16.1	8	+ 8.3±3.6	16.4	14	+6.1±0.6	10.3
80–100	6	+1.7±1.8	19.5	7	+10.6±4.7	16.2	11	+6.0±1.2	12.3
110–130	6	−1.7±1.4	23.8	8	− 1.8±0.8	19.5			
140–160	5	0	26.7						

TABLE II

Mean deviation in blood sugar in patients who received thiopentone–nitrous oxide–oxygen anaesthesia, with and without d-tubocurarine chloride and/or pethidine hydrochloride.

Adjuvant	Mean control blood sugar mg/100 ml	Time in minutes from induction	Number of observations	Mean deviation in blood sugar mg/100 ml	Mean dosage (mg/kg)		
					Thiopentone	d-Tubocurarine chloride	Pethidine
Nil	98	0	14	—	—	—	—
		20– 40	14	+4.0±1.6	7.9	—	—
		50– 70	14	+6.1±0.6	10.3	—	—
		80–100	11	+6.0±1.2	12.3	—	—
d-Tubocurarine chloride	83	0	15	—	—	—	—
		20– 40	15	+3.0±1.1	5.3	0.38	—
		50– 70	12	+7.3±1.4	6.7	0.43	—
		80–100	10	+9.5±1.8	7.4	0.44	—
Pethidine	96	0	12	—	—	—	—
		20– 40	12	−4.0±2.2	7.5	—	0.57
		50– 70	12	−3.0±2.3	10.0	—	0.84
		80–100	12	0	11.1	—	0.99
d-Tubocurarine chloride and pethidine	90	0	10	—	—	—	—
		20– 40	10	+1.8±1.4	7.4	0.34	0.43
		50– 70	10	+2.8±1.8	8.7	0.35	0.58
		80–100	8	+5.4±1.8	9.1	0.37	0.67

atropine cases. This was not due to any particular synergism between promethazine and thiopentone, but to the fact that these were the more recent cases, and it is now appreciated that satisfactory operating conditions can be obtained with smaller doses of thiobarbiturates (Dundee, Price and Dripps, 1956). However, the doses used in the morphia–atropine series were similar to those used in the controls, where no hyperglycaemia was found, and factors other than dosage of thiobarbiturate must have been responsible for the differences observed with the two types of preoperative medication.

Adjuvants.

In this study all the patients received 25 mg of promethazine and 0.6 mg of atropine, approximately one hour before induction of anaesthesia.

Table II shows the changes in blood sugar when d-tubocurarine chloride (dtc) and/or pethidine were used as adjuvants to thiopentone–nitrous oxide–oxygen anaesthesia. These are compared with data from cases who received the thiopentone–nitrous oxide–oxygen mixture alone. Respiration was mechanically controlled in patients who received the relaxant and a closed circuit technique was used. In the other cases spontaneous respiration with a semi-open circuit was used. Although the nature of the operations to be performed differed in cases receiving dtc from those who were not given a relaxant, as far as this study is concerned, they can be considered to be body surface procedures. In all cases anaesthesia was induced well in advance of the onset of surgery and no estimations were made after opening the peritoneum.

Thiopentone–nitrous oxide–oxygen–dtc caused a small, but statistically significant, rise in the blood sugar which increased with the duration of anaesthesia. However, allowing for the error in determination of blood sugar, this was no greater than in the patients who received no relaxant. In none of the patients who received pethidine, was there any significant change in the blood sugar.

Glucose tolerance.

These determinations differed from those in the previous study (Dundee, 1956) in that they were carried out under clinical conditions in subjects undergoing or about to undergo a wide variety of operations. The rate of infusion of glucose was constant throughout—100 ml of 5 per cent solution per half hour. The infusion was started within a few minutes of induction of anaesthesia. Blood samples were taken as near as possible to every half hour. An obvious precaution was to ensure that venous blood was never sampled from the arm used for the infusion.

Control readings, without anaesthesia, were made on patients who were premedicated and prepared for operation exactly as in the anaesthetized cases.

The findings with thiopentone–nitrous oxide–oxygen with and without dtc do not differ significantly from those in conscious subjects (table III). For the sake of clarity this is shown graphically in figure 1. Although there was a slightly greater degree of hyperglycaemia in the anaesthetized patients, this was never statistically greater than in the controls.

In the curarized patients, the final blood sugar reading was actually less than that obtained 30 minutes previously, even though the dextrose infusion was continued. In these seven patients, intra-abdominal procedures were in progress when the last blood sample was drawn and stimulation of the vagus, with consequent outpouring of acetylcholine (Wright, 1952) may be the cause of this apparently anomalous finding.

DISCUSSION

This study illustrates the fallacy of applying purely experimental findings to routine clinical practice. Although it has been shown that thiopentone interferes with the handling of an excess load of glucose, this evidence was obtained with doses of glucose greatly in excess of those employed during a routine infusion. With clinical doses of glucose, even though there may be some impairment of the glycogenolytic-glycogenic activity of the liver by thiopentone, the effect is so small as to be of no clinical significance in fit subjects. It may, however, be of some importance in diabetics who are not stabilized, but even in them clinical experience seems to indicate that the thiopentone–nitrous oxide–oxygen sequence, with or without dtc and/or pethidine, is as safe as any other technique of general anaesthesia

TABLE III

Blood sugar changes during infusion of 5 per cent dextrose at a constant rate, in conscious and anaesthetized subjects.

Anaesthesia	Mean control blood sugar mg/100 ml	Time in minutes from induction	Number of observations	Mean deviation in blood sugar mg/100 ml	Mean dose of thiopentone		Mean dose of d-tubocurarine chloride	
					mg	mg/kg	mg	mg/kg
Nil	96	0	14	—				
		25– 35	14	+28±3.1				
		55– 65	12	+42±5.6				
		85–195	10	+54±3.2				
Thiopentone–N_2O–O_2	97	0	14	—	—	—		
		25– 35	14	+38±6.0	600	9.3		
		55– 65	12	+49±7.3	740	12.1		
		85– 95	6	+52±4.6	860	12.1		
Thiopentone–N_2O–O_2–d-Tubocurarine chloride	98	0	18	—	—	—	—	—
		25– 35	18	+34±2.6	420	7.0	31	0.52
		55– 65	15	+47±6.0	567	9.3	38	0.63
		85– 95	10	+62±5.8	521	8.3	44	0.69
		115–125	7	+61±7.5	553	8.5	46	0.71

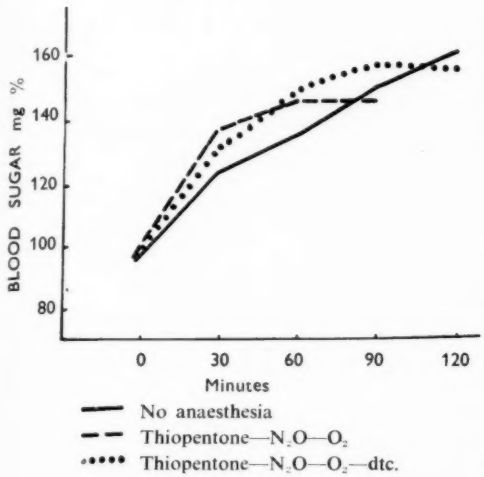

FIG. 1
Graphic representation of table III.

available today, provided hypoxia and hypercarbia do not occur. The importance of these latter two factors in causing hyperglycaemia is shown when the effects of morphia and promethazine are compared (table I) as premedicant drugs. Although it has never been proven conclusively, many authors refer to the fact that promethazine is much less potent a respiratory depressant than the opiates (Hopkin et al., 1957).

McIntyre (1947) listed sixteen references to the hyperglycaemic action of curare. All this work was done with crude curare preparations using various animals. Four of these authors suggested asphyxia as the cause of the rise in blood sugar, two stated that it was not due to asphyxia, while the remainder made no mention of the effects of the drug on respiration. Histamine release, causing liberation of adrenaline is a possible cause of hyperglycaemia following dtc, but Macintosh and Paton (1949) have shown that 0.6 mg/kg of dtc was the smallest dose required to produce this effect. It is suggested from the results of the present study that the hyperglycaemic action of dtc is so small as to be of no clinical significance.

Ward and Dance (1957) have shown that suxamethonium, like dtc, has minimal effects on blood sugar. One must add to these statements the important provisos listed by Foster and Francis: "... provided that (*a*) severe falls in blood pressure are avoided, especially during induction with thiopentone, (*b*) anoxia and carbon dioxide retention are at all times avoided, and (*c*) adequate depth of anaesthesia is maintained to prevent sympathetic stimulation (sweating, pallor, etc.)."

SUMMARY AND CONCLUSIONS

Thiopentone–nitrous oxide–oxygen anaesthesia with various forms of pre-operative medication and with various adjuvants, has been studied with reference to its effects on blood sugar and glucose tolerance. It was found that:

(1) Except where morphia and atropine was used for pre-operative medication, the thiopentone–nitrous oxide–oxygen sequence did not produce any appreciable degree of hyperglycaemia. The changes observed with morphia premedication are probably due to the respiratory depression which it produces when given prior to thiopentone.

(2) Neither d-tubocurarine chloride nor pethidine aggravates the effects of the thiopentone combination on blood sugar, provided adequate respiratory exchange and carbon dioxide elimination are maintained.

(3) Under clinical conditions the effects of thiopentone–nitrous oxide–oxygen, with or without d-tubocurarine chloride and/or pethidine, on glucose tolerance are so slight as not to increase appreciably the hyperglycaemia produced by a slow infusion of 5 per cent dextrose.

The fallacies of applying the findings of laboratory investigations to routine anaesthesia practice are discussed.

ACKNOWLEDGMENTS

Part of this data was published by permission of the editor of the *British Journal of Pharmacology and Chemotherapy*.

REFERENCES

Dundee, J. W. (1956). Effect of thiopentone on blood sugar and glucose tolerance. *Brit. J. Pharmacol.*, **11**, 458.

—— Price, H. L., and Dripps, R. D. (1956). Acute tolerance to thiopentone in man. *Brit. J. Anaesth.*, **28**, 344.

Foster, P. A., and Francis, B. G. (1955). An Operation for the diabetic? *Brit. J. Anaesth.*, **27**, 291.

Goldsmith, M. W., and Holmes, F. (1957). Thiopentone and carbohydrate metabolism. *Anaesthesia*, **12**, 321.

Hawk, P. B., Oser, B. L., and Summerson, W. H. (1947). *Practical Physiological Chemistry*, 12th ed., p. 520. London: Churchill.

Hopkin, D. A. B., Hunter, D., and Jones, C. M. (1957). Promethazine and pethidine in anaesthesia. *Anaesthesia*, **12**, 276.

Macintosh, F. C., and Paton, W. D. M. (1949). The liberation of histamine by certain organic bases. *J. Physiol.*, **109**, 190.

McIntyre, A. R. (1947). *Curare—its History, Nature and Clinical Use*, p. 142. Chicago: University of Chicago Press.

Ward, R. J., and Dance, C. L. (1957). Blood sugar changes following the administration of succinylcholine. *Curr. Res. Anesth.*, **36**, 65.

Wright, S. (1952). *Applied Physiology*, 9th ed., p. 917. London: Oxford University Press.

BOOK REVIEW

Anesthesia and Otolaryngology. By Donald F. Proctor. Published by Baillière, Tindall and Cox, London. Pp. 267. Price 56*s*.

Ear, nose and throat surgeons have always taken the closest interest in anaesthesia. Their anatomical field of work overlaps that of the anaesthetist, and neither worker can do his best if the other is inexpert. More than one anaesthetist can remember a kindly E.N.T. surgeon getting him out of difficulty by passing an endotracheal tube for him. Few anaesthetists of experience have not at some time or other murmured helpful words of advice to young E.N.T. surgeons whose enthusiasm has outstripped their experience.

Dr. Proctor, an E.N.T. surgeon of 15 years standing, was an anaesthetist at the John Hopkins Medical School for four years. He writes from the standpoint of the E.N.T. surgeon instructing in anaesthesia. There are no doubt surgical colleagues of Dr. Proctor in many parts of the world who cannot call upon the services of a specially trained anaesthetist. They will find his book an admirable and in the main reliable account of anaesthesia for their branch of surgery. They can cull sufficient from it to oversee the work of their nurse or orderly, or even to administer the anaesthetic themselves.

Anaesthetists too will read this book with profit. It does set out the opinions of an experienced surgeon and as such are worthy of respect. A particularly valuable section on the use of the local anaesthesia for ear, nose and throat procedures, shows the author at his best. This is a little offset by his spill-over, somewhat unwisely, into the field of thoracic anaesthesia and resuscitation of the newborn.

The book is a mixed bag. E.N.T. surgeons will find it of the greatest value, but should be cautious that their newfound knowledge does not bring them into conflict with their own highly trained anaesthetists. Anaesthetists will learn a great deal from such snippets of surgery as Proctor gives them, and should respect the sound common sense of the anaesthetic advice even though some of the techniques may be different from those in use in this country. The illustrations are few but helpful. Glossy paper would have made them clearer and more easy to study. Useful bibliographies are appended to the end of each chapter.

W. W. Mushin

CORRESPONDENCE

ERRATUM

Sir,—In spite of great care by all concerned a few minor errors crept into the final copy of my article on "Terminology and Symbols used in Respiratory Physiology" (*Brit. J. Anaesth.*, **29**, 534). None of them is serious, but perhaps those of your readers who wish to make use of the article may like to correct them.

p. 536 TABLE II (IV) : *for* v *read* $\overline{v}$.

p. 538 TABLE IV (V) : *for* respiration quotient *read* respiratory quotient.

TABLE IV (VII): *for* $\frac{P_{AC_{O_2}}}{R}$ *read* $\frac{P_{A_{CO_2}}}{R}$

for (i−R) *read* (I−R).

E. J. M. CAMPBELL
Middlesex Hospital, London, W.1

Brit. J. Anaesth. (1958), **30**, 83

ANAESTHESIA IN RECURRING POST-TONSILLECTOMY HAEMORRHAGE DUE TO MACROGLOSSIA

A Case Report

BY

P. F. KNIGHT

Cambridge Military Hospital, Aldershot.

THE management of anaesthesia for control of haemorrhage following tonsillectomy constitutes a problem fraught with well known hazards, in a patient often shocked and usually with an airway partially obstructed by blood and clot in the pharynx. The danger of blood entering the trachea during induction has resulted in the widely held view that the anaesthetic agents of choice should be gaseous or volatile, with spontaneous respiration and an initially active cough reflex.

The case here described is of unusual interest on several counts. The patient was a mentally retarded child with macroglossia and other congenital abnormalities, which resulted in a postoperative cycle of respiratory obstruction associated with struggling and venous congestion causing repeated exacerbations of haemorrhage. This failed to respond to the usual local haemostatic measures, and, ultimately, tracheostomy proved the only means of breaking this circle of events. In addition it was found necessary to employ the intravenous route for the four further anaesthetics administered to the child in the twenty-four hours following tonsillectomy.

CASE REPORT

A male child aged five years was admitted to the Louise Margaret Hospital, Aldershot, for routine tonsillectomy. It had been noted at previous outpatient attendance that the child was slow and awkward of speech and appeared mentally retarded, and the mother had mentioned its "difficulty in breathing" since birth.

When seen pre-operatively by the anaesthetist, the child was of normal build for its age, but was very restless and obviously mentally subnormal. There was a marked funnel-shaped deformity of the sternum and a receding lower jaw, while inspection of the oropharynx revealed macroglossia, a high arched palate and large fleshy tonsils. Even at rest mouth breathing was stertorous, but the child was a good colour and otherwise well.

Premedication was with 135 mg (2¼ grains) of pentobarbitone sodium and 0.65 mg of atropine given two and one hours before operation, with satisfactory effect. Anaesthesia was induced under an open mask with ethyl chloride, and slight difficulty was experienced owing to obstruction by the tongue, until an oral airway could be introduced and was tolerated. After deepening anaesthesia with open drop ether, maintenance was effected with nitrous oxide–oxygen (5 l.: 3 l.) and ether delivered via a Boyle-Davis gag, and was smooth and uneventful. After dissection tonsillectomy and adenoidectomy had been performed the child was returned to the ward with an oral airway in situ and breathing freely.

Four hours later the child was reported to be bleeding from the mouth and nose, and when seen was pale and obviously shocked and had a blood pressure of 70/40 mm Hg. There was a steady trickle of blood from the mouth and a lot of clot in the pharynx. Although "round" from the anaesthetic he was breathing with some difficulty and swallowing blood. After taking blood for cross-matching, an intravenous infusion of dextran was started and the child taken to the theatre, having had 0.3 mg of atropine intravenously.

Induction of anaesthesia was attempted in the Trendelenburg position after sucking as much blood as possible from the posterior pharynx; but the use of gas and ether for induction proved well nigh impossible since the large tongue and blood clot constituted an almost total obstruction as soon as consciousness was lost, while the jaw was too tightly clenched to allow introduction of a gag or airway. As a result a gaseous induction was abandoned and thiopentone 150 mg given into the drip tubing, followed by 40 mg of suxamethonium. As soon as the jaw relaxed, and, without inflating the lungs, laryngoscopy was performed and a great deal of blood and clot sucked from the pharynx. The child was then intubated with a plain oral Magill tube and the larynx packed off—anaesthesia being maintained with gas, oxygen and ether given through an Ayres T-piece. Small bleeding points in both tonsillar fossae were ligated, and the residual ooze responded to packing. During operation the child received 500 ml dextran and 250 ml of blood, and after extubation he vomited about 750 ml of altered blood. He returned to the ward however, in fairly good condition and with the blood drip still running.

After about two and a half hours it was reported

that the child was bleeding again. When seen his airway was obviously obstructed and he was struggling for breath, with active accessory muscles, cyanosis and gross venous congestion of the neck and face. The abnormally large tongue coupled with faucial oedema from previous manipulations proved resistant to attempts at maintaining an airway by jaw traction, and the bleeding continued despite relief of the obstruction by use of tongue forceps. Three hours after the previous anaesthetic, and after further intravenous premedication with 0.65 mg of atropine the child was taken to the theatre once more. In view of the previous experience, anaesthesia was induced by thiopentone 100 mg followed by 40 mg of suxamethonium given with the patient in the Trendelenburg position, and followed by oral intubation under direct vision after clearing the pharynx of blood. Maintenance was again smooth with gas, oxygen and ether given via a T-piece. Haemorrhage was still profuse, being mainly from the postnasal space, and was controlled only by a pack tied into position. After the second attempt at haemostasis, the child was once more returned to the ward with the airway in situ and a slow blood drip running; his general state being fairly good.

Less than six hours later, however, he was gagging on the tape retaining the postnasal pack in place, and was again semi-obstructed with grossly congested face and neck; and, as a result of these conditions, haemorrhage recommenced.

It was by now apparent that each time the child recovered consciousness enough to be unable to tolerate an airway, its overlarge tongue (now rendered even larger at its base by oedema) caused severe respiratory obstruction with consequent venous congestion and hypercarbia, which led inevitably to renewed haemorrhage from all raw areas in the pharynx. By this time 1 litre of blood and 500 ml of dextran had been given. It was therefore decided to perform temporary tracheostomy and this was done about fourteen hours after the original tonsillectomy. The method of anaesthesia used was identical with that employed on the previous two occasions, and again haemostasis in the pharynx was effected by packing.

The provision of a clear airway proved finally successful in preventing further episodes of haemorrhage, and on the following day the postnasal pack was removed under nitrous oxide anaesthesia administered through the tracheostomy tube via a T-piece.

Somewhat remarkably, in view of the five anaesthetics in under thirty hours, the child suffered no further complication, and after subsidence of its faucial oedema and removal of the tracheostomy tube two days later, was able to breathe quite freely.

When seen at a later date the stoma had healed cleanly with no obvious tracheal stenosis.

COMMENT

The notable feature of this case with its associated problems, was the relative ease and smoothness with which anaesthesia was established under difficult circumstances, by the use of an intravenous induction and a short-acting relaxant. It is felt that provided a strong sucker is available and fairly steep Trendelenburg used, the risk of blood passing into the trachea is negligible. Further, it was evident that, even without inflating the lungs after induction, there was less risk of anoxia occurring in the short time required to clear the pharynx of blood and perform intubation, than there was with a gas or ethyl chloride induction, with the struggling, coughing, and increased bleeding that are only too often associated.

The use of a 2.5 per cent solution of thiopentone in a mildly shocked child did not produce severe hypotension, and was perfectly satisfactory while the employment of an Ayres T-tube and a fairly high gas flow reduced expiratory resistance to a minimum and prevented carbon dioxide accumulation.

In view of the experience in this case the desirability of avoiding surgery of the nose or pharynx in similar subjects, unless absolutely essential, seems clearly indicated.

SUMMARY

A case is recorded of macroglossia in a mentally retarded child, leading to repeated episodes of haemorrhage following tonsillectomy. The anaesthetic management of this problem is discussed, and the advantages experienced from the use of an intravenous induction plus a short-acting relaxant and intubation are mentioned.

ACKNOWLEDGMENT

I am indebted to Lieut.-Colonel A. C. Cox, O.B.E., R.A.M.C., the otorhinolaryngologist concerned, for permission to publish this case.

Brit. J. Anaesth. (1958), **30**, 85

DEATHS UNDER AN ANAESTHETIC*

A Critical Review of the Procedure and Consequences of Reporting such Incidents

BY

ROBERT LAWRIE

County and City of Perth General Hospitals, Scotland

INTRODUCTION

LARGELY because of the advances in anaesthesia, many surgical procedures are now undertaken which formerly were considered, if not impossible, at least unjustifiable, either because of the nature of the operation or the condition of the patient. This has led to a situation in which the possibility of a patient dying in association with an anaesthetic has increased. In many of these cases the anaesthetic may be merely an incident. In addition, and possibly because of the desire for free and open discussion, there appears to have been an increasing amount of publicity over deaths associated with anaesthesia or matters connected with it (*B.M.J.*, 1956 a, b; Brotherton, 1956). One cannot help feeling, therefore, that the casual observer may be left with impressions which are misleading. In Scotland a factor which influences these impressions, is the choice of the words "death under an anaesthetic" to indicate a set of circumstances requiring Fiscal investigation, and which, by common usage, have come to be referred to as "anaesthetic deaths." Either term conveys implications which may be misleading.

The interpretation of the terms and the legal procedure appear to vary considerably, if one can judge from the opinions expressed by all grades of anaesthetic and surgical staff who arrive from time to time in our own group of hospitals. Some of these interpretations seem to be quite unreasonable. I propose, therefore, to survey this aspect of the subject, firstly by describing an incident which occurred recently, secondly by enquiring into the formalities of the legal procedure, and finally by briefly considering some of the long term results of the initial reporting, with particular reference to the Vital Statistics.

A RECENT INCIDENT

The incident concerned a little girl aged 2, who was taken to the casualty department of a neighbouring infirmary. She had fallen on a bamboo cane and had a small incised wound of the neck, which was examined and sutured by the casualty officer. She was sent home, and 2 hours later was admitted in a critical condition, with an increasing surgical emphysema of the neck and chest wall, respiratory distress and shock. It was then appreciated from all the circumstances, that the child had fallen out of a back window, a height of about 4 feet 6 inches, had impaled herself through the neck, and the cane had in fact penetrated the chest. A thoracotomy was commenced about 11 p.m. The resident anaesthetist had called in the duty consultant: the child was anaesthetized with cyclopropane, intubated under suxamethonium relaxation. Throughout the operation the child received infinitesimal amounts of cyclopropane. The thoracotomy revealed an extensive tear of the oesophagus, with spillage and contamination of the mediastinum. This was repaired, and for a period following this and transfusion, the child's condition appeared to improve, though this was only relative. The operation was completed in approximately 2 hours, and at 1.30 a.m. the consultant anaesthetist, the resident anaesthetist, the surgeon and his assistant were all satisfied that the child was sufficiently recovered from the anaesthetic to allow of her return to the ward. She was only 2 years old, was still critically and dangerously ill, but she moved her limbs actively, resented any form of artificial airway, and opened her eyes. The child was placed in an

* Modified from the Presidential Address to the Scottish Society of Anaesthetists, April 1957.

oxygen tent, the i.v. fluid continued, and her care was entrusted to the house surgeon, who had *not* been present throughout the operation, or at the close of operation, but had been in and out of theatre and had been preparing for her reception in the ward. The child died approximately 6 hours later. The house surgeon, without informing anaesthetist or surgeon, reported the death as a death under an anaesthetic and as a fatal accident, doing so via the medical superintendent to the Procurator. He did so because the last time he had seen the child alive was while adjusting the i.v. drip, and, as the child had not responded to this stimulus, he assumed that when she later died she had been still unconscious as a result of the anaesthetic, and that anyway he was under the impression that all cases dying within 24 hours following an anaesthetic should be reported to the Fiscal as a death under anaesthesia. The Fiscal sent the police surgeon's deputy to report, and she certified the death as follows: "Death after accident due to shock while under anaesthetic for thoracotomy for rupture of the oesophagus, the anaesthetic being curare, cyclopropane, oxygen and Prostigmine." The anaesthetist refused to complete the report form normally used in the infirmary concerned, on the grounds that this case did not fall into the category of a death under an anaesthetic. Both the surgeon and the anaesthetist objected to the phrasing of the death certificate, and sent a written protest to the Fiscal, stating that the certificate was false and untrue in their opinion.

The Crown Office instructed that a Fatal Accident Enquiry be held, and this enquiry in the public court was later conducted by the Sheriff, before a jury of seven. It is possible that this enquiry took place solely because of the disputed terms of the death certificate, and the Fiscal himself seemed to think this was in fact the reason for the enquiry. It happened, however, that by a strange coincidence, a second case involving anaesthetics was held in the same court that morning, and it is also possible that this may have been brought about partly by the fact that legal attention had been unduly focused on the case I have described.

I attended the Sheriff's enquiry. It would take too long to describe the entire proceedings but I would like to bring out some of the points which struck me as significant.

(1) That the house surgeon and the pathologist both stated that because the patient had not fully recovered consciousness before her death, and also because death had occurred within 24 hours following an anaesthetic, they were under the impression that it should be regarded as a death under anaesthetic, as this, they thought, was common practice.

(2) That the deputy police surgeon, even under pressure from the Sheriff and the Procurator, persisted that because she had been told that the child had not fully recovered consciousness before she died, it could not be said that the child was not under the influence of the anaesthetic and should therefore be regarded as a death under an anaesthetic. She persisted with this even when it was suggested that the child may have lapsed into unconsciousness because of shock, following a period of temporary recovery.

(3) That the pathologist, and the police surgeon's deputy, made it quite clear that though they regarded it as a death under an anaesthetic, the anaesthetic was not the cause of death and may not have contributed at all.

(4) That the Sheriff conducting the enquiry was mainly interested in establishing this fact and the cause of death, and that due care had been shown by all concerned.

(5) That despite the obvious severity of the injuries and the other circumstances of the case, all the limelight was on the anaesthetic.

The Sheriff summing up the case said that the cause of death must inevitably be "Shock following an accident", and verdict should be "Died following shock". He made no mention of the anaesthetic in the cause of death, however, and this seemed to disturb the Procurator, who was most insistent on the exact recording of the details on the death certificate, and quoting a medical jurisprudence textbook, used the words " 'following the administration of an anaesthetic' such cases should be recorded". To which the Sheriff replied that if that was intended to be taken literally, his own death certificate should eventually contain the words "following an anaesthetic", as he had, in fact, had a general anaesthetic at the age of 13 for appendicectomy!

This particular case was undoubtedly an excep-

tional one, inasmuch as it is probable that the anaesthetist's objection to the phrasing of the death certificate resulted in a public enquiry. All of us, I am sure, must have experienced a similar state of affairs, in cases which do not survive operation for many hours, when the question of reporting the death to the Fiscal arises. Invariably, one hears a variety of interpretations of the term "death under an anaesthetic", and equally invariably the term becomes from convenience "anaesthetic death". Even in this particular case the terms "death under anaesthetic" and "anaesthetic death" were used synonymously in the public court. Invariably, it always seems, unnecessary emphasis is placed on the part played by the anaesthetic. It is only a short step from this to the emphasis being placed on the anaesthetist if he is unlucky enough to be involved in a series and likewise also on one particular anaesthetic. When all the circumstances of each particular case have been forgotten, the summation of all these effects may conceivably influence any single anaesthetist, not only in his choice of anaesthesia, but even in his decision to anaesthetize a poor risk patient at all. It is important, therefore, that we know at least what is our public responsibility in this matter, and I propose now to examine this from the official information available.

OUR LEGAL RESPONSIBILITY

There is no *specific* law concerning "deaths under an anaesthetic". The Procurator Fiscal's enquiry into such cases falls into the general category covered by the Fatal Accidents and Sudden Deaths Inquiry Acts of 1895 and 1906. The duties of the Procurator Fiscal in such cases is to eliminate possible crime in all its degrees, including negligence or undue lack of care and in cases of doubt, the cause of death. Without quoting chapter and verse in detail, which would take too long, I am assured that this is in essence correct. In view of the circumstances which may surround a death under an anaesthetic, however, possibly because such deaths constitute a considerable proportion of deaths from other than natural causes, the Lord Advocate has issued from time to time certain directions to hospital and institutional authorities. The latest of these, in the form of a circular letter dated April 1938, is as follows:

CROWN OFFICE,
EDINBURGH, 1.
April 14, 1938

Sir/Madam,

DEATHS UNDER ANAESTHETICS

The reporting of deaths occurring while patients are under the influence of an anaesthetic has been under further consideration of the Crown Authorities with a view to clearing up doubts raised by certain Institutional Officials.

As a result, the Lord Advocate has instructed me to direct your attention to the matter and to advise you that the necessity for reporting such deaths to the Procurator Fiscal applies to all cases where death occurs during anaesthesia whether in the Operating Theatre or after removal to the ward or room.

This applies to cases so occurring even although the explanation of the death may be clearly attributable to the condition of the patient apart altogether from any question of an anaesthetic factor.

Such cases fall to be reported whether the anaesthetic be a general anaesthetic administered by inhalation or by injection into the blood stream (i.e. evipan) or spinal canal (i.e. spinal anaesthetic drugs) or per rectum (i.e. avertin) or be cases of some combination of such drugs with local anaesthesia.

So too cases of deaths occurring while the patient is under the influence of a local anaesthetic fall to be reported to the Procurator Fiscal.

I am, Your Obedient Servant,
(*Sgd.*) R. H. JAMES,
Crown Agent.

The letter is somewhat old, but is sufficiently widely phrased to cover all circumstances in the present-day theatre. From perusal of this letter, therefore, there are only two points at issue.

(1) What constitutes (*a*) under an anaesthetic; (*b*) under the influence of an anaesthetic; or (*c*) during anaesthesia?

(2) What constitutes a report?

To refer to the terms under, under the influence of, or during anaesthesia, there is very wisely no attempt at exact definition. The decision concerning this is entirely up to the medical authorities reporting the case and would, of course, vary with individual circumstances.

It is important here to remember that the Fiscal's interest in these cases is to eliminate possible crime by enquiring into the circumstances of the death. It is *not* a scientific or clinical investigation as we think of it. It is entirely to safeguard the public interest with reference to any particular case or even series of cases. It is when we attempt to put exact definitions to the terms that difficulties arise.

If some level of consciousness is used as a criterion, then what level? How do we assess whether unconsciousness is due to the anaesthetic, to a pre-existing cause as in the head injury, or to an intervening cause such as shock in the case of the child I described. If we try to be scientific in defining the term "under the influence", then what concentrations and of which drugs do we consider exert sufficient influence to fall into this category? If some reference to time is included in any definition, it must be an entirely artificial one. It *may* be reasonable enough to report to the Procurator every case which dies within 24 hours of an anaesthetic: it is completely unrealistic to report every such case as a death under an anaesthetic.

If, on the other hand, one attempts to be explanatory, as for example in Glaister's *Medical Jurisprudence*, and use such phrases as "following an anaesthetic", it may be quoted verbatim and out of context again to the point of absurdity. Obviously each individual case must be assessed on its merits, using one's common sense, bearing in mind the essential reason for the necessity of reporting such cases, namely to allow the Procurator Fiscal to enquire into all the circumstances of any sudden death not due to natural causes, so that he may exclude criminality. It is important, therefore, that he must be presented with a reasonable, unbiased picture of the circumstances. To adhere to any rigid definition of the term "under an anaesthetic" to cover all cases, cannot possibly allow a reasonable picture to be conveyed in view of all the possible factors involved in any one case. There will be errors of both omission and commission.

The second point at issue is—what constitutes a report?

In addition to the variations in the interpretation of the words "death under an anaesthetic", there appear to be various interpretations of the manner in which these cases are initially reported to the Fiscal.

In our own area, for example, we simply inform the Fiscal by telephone, and as a result of this, the police surgeon is sent to investigate, and he then completes his own report with particular attention to points asked by the Fiscal. There is, however, no formal or official document used in either case.

The procedure outlined by Glaister (1953) in his book, however, advises that with the announcement to the Fiscal a report by the patient's doctor is furnished on *a form* supplied from the office of the Fiscal. In this form all the emphasis is placed on the anaesthetic and when it is completed it is given to the medico-legal investigator, usually the police surgeon, to enable him to complete his report. Such a form or similar forms are in use, I know, in certain parts of Scotland. I have distributed a specimen copy of one of these (see page 89).

It would seem, however, that, apart from the emphasis laid on the anaesthetic details in this report, any printed form which is specifically headed "Report as to the Death of So-and-so under an Anaesthetic" predetermines the way in which the investigating medical practitioner will make his report to the Procurator Fiscal. It cannot allow for the case in which there may be any doubt in the matter, and this will be further emphasized if a rigid questionnaire is adhered to, which does not allow of a full description of all the circumstances. When I first heard of the fact that such a report form existed, I must say I felt rather worried, because perhaps we had omitted something in our own hospitals. I am assured, however, by both our own Procurator Fiscal and the Crown Agent's Office that the method of the initial reporting of a case is entirely one of local arrangement, and that although such forms may be used, they are in no way statutory forms, and a simple statement over the telephone is quite in order. I find it difficult to estimate just how important the manner of reporting these cases may be, but if one remembers that it is under the general category of all sudden deaths that we report "deaths under an anaesthetic", the method and phraseology we use would seem to be of importance. It should also be remembered that while it is our responsibility to report such cases to the Procurator, it does not necessarily mean that they must inevitably be certified as such. Any printed form, however, on the lines shown, is more likely to lead to this happening.

THE PROCURATOR'S RESPONSIBILITY

In order that we can more clearly understand the procedure which happens after a case has been reported to the Procurator, I asked for, and was

[COPY]

REPORT BY.....

of ..... as to the death of

.....

while under anaesthetic for operation.

WARD No.....

(1) Full Name of Patient .....

(2) Age..... (3) Address.....

.....

.....

(4) When admitted to hospital.....

(5) Nature of Disease or Ailment.....

(6) Date of Operation and hour of death and opinion whether death was due primarily to respiratory or heart failure:

.....

(7) When and by whom was patient informed anaesthetic necessary?

.....

(8) What precautions were used in preparing patient for its administration?

.....

.....

(9) When and by whom was patient examined as to condition of heart, lungs and urine and what were their condition?

.....

.....

(10) Anaesthetist: Anaesthetic: Method of Administration:
Time during which patient was under the influence of the Anaesthetic?

.....

.....

(11) What Surgeon and Physicians were present at operation?

.....

(12) Who was Operator?.....

(Signature).....

(Qualifications).....

kindly given, permission to copy and have circulated the Regulations for Procurators Fiscal from the Crown Advocate on the whole subject.

[COPY]

PROCURATOR FISCAL SERVICE, 1945

CROWN OFFICE EDINBURGH

REGULATIONS TO BE OBSERVED IN CRIMINAL AND OTHER INVESTIGATIONS

Section SUDDEN DEATHS

Para. 10

Death under Anaesthetic.

In all cases of deaths under chloroform or any other anaesthetic, administered by a medical man with a view to an operation or otherwise, Procurators Fiscal will obtain a report from an independent medical man, not only on the cause of death, but also as to whether all proper precautions were observed in the administration of the anaesthetic. To enable the independent medical man to prepare his report, Procurators Fiscal will submit to him the precognitions taken from persons present when the anaesthetic was administered and ask him to direct particular attention to the following points, viz.:

(1) Whether the patient was medically examined before it was decided that an anaesthetic should be administered.

(2) Whether all due precautions were observed in the actual administration of the anaesthetic.

(3) Whether there were any symptoms about the patient which might or ought to have been discovered by examination, indicating that the administration of an anaesthetic would be attended with special risk to life.

To enable such a report to be completed it will in some cases be necessary for the independent medical man, when making his examination, to dissect the body, but it is not to be understood that dissection is necessarily to be resorted to in all cases.

If the independent medical man is satisfied that the administration of the anaesthetic was advisable and that every proper precaution was taken before it was administered, he will probably find it unnecessary to make a dissection. The expediency of doing so or not doing so, is left entirely to his discretion.

The attention of Procurators Fiscal is drawn to Circular Letters directed by the Lord Advocate to Hospitals and Nursing Homes: (1) dated 28th September, 1925, drawing attention to (*a*) the danger to life which may occur from chloroform being administered in mistake for ether during anaesthesia, and (*b*) recommending that these liquids should always be stored in bottles distinctly coloured and clearly labelled; and (2) dated 3rd December, 1931, regarding the danger of allowing medical students to administer anaesthetics in which the administration of the anaesthetic is not under supervision and recommending that in no case should any unqualified medical person be permitted to administer an anaesthetic except in the presence of a qualified medical practitioner, and under his immediate supervision; and (3) dated 14th April, 1938, advising Hospital Authorities as to the necessity of reporting deaths to Procurators Fiscal in respect of all cases where deaths occur *during* anaesthesia whether in the operating theatre or after removal to the ward or room and also (*a*) that this direction applies to cases even although the explanation of the death may be clearly attributable to the condition of the patient apart altogether from any question of an anaesthetic factor, (*b*) that such cases fall to be reported whether the anaesthetic be a general anaesthetic administered by inhalation, by injection into the blood stream (i.e. evipan) or spinal canal (i.e. spinal anaesthetic drugs) or per rectum (i.e. avertin) or be cases of some combination of such drugs with local anaesthesia, and (*c*) that all cases of deaths where the patient is under the influence of a local anaesthetic fall to be reported to the Procurator Fiscal.

Procurators Fiscal will report to the Crown Office if there is any failure to comply with the terms of the letter.

Without going into too much detail, I would ask you to note the following points:

(*a*) That the person delegated by the Procurator Fiscal to investigate the circumstances of these cases is described as an independent medical man: he is, therefore, not necessarily either an expert in medical jurisprudence or in anaesthesia. He will, therefore, turn to the Procurator for advice on the legal aspects of the case, and if he has specifically been informed by the Procurator that this is a death under an anaesthetic, he must obviously consider it as such and almost inevitably include some such words in his certified cause of death. Even if he is a police surgeon and has special medico-legal knowledge, he must turn to the hospital specialists for their advice on the clinical aspect. Again, if all the emphasis is on the anaesthetic, it would seem highly unlikely that he will not mention this in his certified cause of death and in his report to the Fiscal.

(*b*) That the Fiscal, *having been informed* that this is a death under an anaesthetic, asks the examining medical practitioner to pay particular attention to these three points:

(i) Whether the patient was medically examined before it was decided that an anaesthetic should be administered.

(ii) Whether all due precautions were observed in the actual administration of the anaesthetic.

(iii) Whether there were any symptoms about the patient which might or ought to have been discovered by examination, indicating that the administration of an anaes-

thetic would be attended with special risk to life.

Again, all the emphasis is on the anaesthetic, and in these questions, the anaesthetist. This is inevitable in view of the nature of the information the Procurator has been given in the first place by the initial report.

It would, therefore, appear to be of considerable importance how we inform the Procurator and in what words. If there is any doubt about the patient being under an anaesthetic, then I see no reason why it should be specifically reported as such: it is still open to report the case to the Procurator as a sudden death or at least a death from other than natural causes, and the whole circumstances of the case can be reviewed in the right perspective.

Before I leave the Regulations for Procurators, it is significant that the letter to the hospital authorities from the Crown Advocate, which I mentioned earlier, has been carefully copied word for word and there is therefore no conflict between the choice of terms in the two sets of instructions.

I have attempted up to now to deal with the immediate legal aspects of these cases. I have, however, always been interested to know what happened next, and I pursued the matter further. I think the results must interest all of us.

THE REGISTRATION OF THE DEATH

With the information given him by the Procurator, the independent medical practitioner (usually the police surgeon, but not necessarily so) investigates the case. He then completes his report to the Procurator, bringing out all the specific points he has been asked, giving his opinion on whether or not he is satisfied that due care has been shown. He states the cause of death and will usually also add some reference to the anaesthetic, and in most cases the nature of the anaesthetic. He will usually do so, because of the emphasis placed on the anaesthetic in his information from the Procurator and any form of written report from the hospital, but he is under no obligation except to indicate fairly the cause or causes of death. At the same time he may write a death certificate or indicate the phrasing of the death certificate, or one may have already been written by the patient's doctor. The death certificate goes to the local registrar and, if this has been received before information is obtained from the Procurator, will be registered in the death register giving the cause of death as written. On receipt of the independent doctor's report and any other evidence, the Procurator completes what is called a "Result of a Precognition", in which he states the cause of death, which usually is the same as that given by the investigating doctor, but which for clarification may be modified, where vague statements have been made.

A copy of this, called a "Schedule for Registrar", is sent to the local registrar and from this the final cause of death is registered. If it disagrees with the cause as already certified, as may happen, the final entry is made in what is called a Register of Corrected Entries. Any future extracts of the death certificate will show the cause as entered originally, with the corrected entry on the back. An extract can be obtained by any one who pleases on payment of a small fee.

The "Result of Precognition", along with any reports and a copy of the schedule for the registrar are sent to the Crown Office in Edinburgh, stating as accurately as possible registration particulars, including the cause or probable cause and circumstances of the death to enable the Crown Counsel to revise the schedule correctly. This procedure is inevitable in all deaths which come into the category of sudden or violent death investigated by the Procurator. The final decision as to how any of these deaths should be registered, rests entirely with the law.

The least we can do then is to present their representatives with a reasonably clear picture of *all* the circumstances which should include the part played by the anaesthetic in its true perspective. To improve the present procedures, however, would present many problems. At the moment we might do well to standardize the form of procedure at present available. I sincerely hope I have said nothing to confuse it further.

One last point as regards the legal side: where death has occurred under an anaesthetic in unusual circumstances or where there are features which suggest negligence, the Procurator Fiscal is instructed to report the case to the Crown Office as soon as possible. Counsel then decide what

steps should be taken, and presumably would first direct a Sheriff's enquiry.

SUMMARY UP TO THIS POINT

To summarize up to this point, therefore:

(1) The responsibility of reporting any sudden or violent death to the Procurator Fiscal is to allow him to investigate the circumstances and exclude criminality in all its degrees.

(2) Because the circumstances surrounding any death under an anaesthetic may conceivably include any degree of criminality, the death must be reported.

(3) Having been reported, the greater the emphasis on the anaesthetic as one of the circumstances, the greater will be the likelihood of the independent doctor and the Fiscal including some reference to the anaesthetic on the registered cause of death.

(4) What starts, therefore, as a circumstance requiring investigation, finishes almost inevitably as a cause or contributory cause of death at least to the lay person who receives a copy of the death certificate which is taken both from the original copy on the death register and from the register of corrected entries.

(5) It would seem highly probable, and almost inevitable, that this would also be the case in the compilation of the vital statistics for the Registrar-General's annual report.

THE STATISTICAL CLASSIFICATION

In order to find out the official interpretation of the particulars in the register, I went to the Register House, where the staff was most helpful and co-operative. At regular intervals they receive from each local registrar, a complete copy of all the entries, including corrected entries, and an indication of whether there has been a Procurator's enquiry.

Each entry is scrutinized and the cause of death classified according to the International Classification of Diseases which has been in general use since 1950. Deaths in which an anaesthetic is involved are classified according to whether the anaesthetic is the primary or contributory cause; if the primary cause it falls into one of two headings:

(1) Therapeutic misadventure in anaesthesia;

(2) Complication of anaesthesia for non-therapeutic purposes.

I mention this difference as it reflects, I think, the influence of the law in this. Non-therapeutic purposes include operations for sterilization, ritual or preventive circumcision, and aesthetic or other purposes.

Before the statistician classifies the anaesthetic as being the primary cause, however, he makes absolutely sure that this is the case; in this and other points he seeks advice from the Chief Statistical Medical Officer in St. Andrew's House. Very few cases are so classified and since the classification has been in use in 1950 it is interesting to note the figures (McKinlay):

Year	1950	1951	1952	1953	1954	1955	1956
No. of cases	6	2	3	2	8	4	0

When I was first given these figures I was highly elated as I thought here at least is an argument that what I personally consider the real anaesthetic deaths, inasmuch as they were due to the anaesthetic, were only a fraction of these spoken of as anaesthetic deaths. Undoubtedly, however, one cannot but feel that these figures are too good to be true, and that in fact the benefit of the doubt is given to the anaesthetist as regards the primary cause at least. This is in fact the case, and the Chief Statistical Medical Officer informed me that the vast majority are classified to the disease or condition for which the operation was performed, the anaesthetic being shown as a contributory factor, but as regards the latter aspect of the classification, the staff at Register House said that if the anaesthetic was mentioned on the death certificate it was included in the classification either as a cause or contributory cause.

I took the opportunity while I was in Register House to see just what was the eventual destiny of the case of the child I have described, and the other case which had been the subject of a Sheriff's enquiry on the same morning at the same court: in this second case there was no doubt that the immediate cause of death was the anaesthetic, the patient in fact died during induction of anaesthesia before operation, although no blame was attached to any individual; the final

certified cause of death in this instance was, by the Sheriff: "Died through the administration of an anaesthetic, and that death was due to myocardial failure."

Both cases were classified according to the condition for which the operation was performed or contemplated as the primary cause, and in both cases the anaesthetic was shown as a contributory cause. Although perhaps the two cases referred to were extremes, they illustrate just how anomalous the situation can be, particularly in view of the fact that in the case of the child, despite the enquiries made specifically because of the wording of the original death certificate, and although the eventual certified cause of death by the Sheriff made no mention of the anaesthetic, the original disputed phrasing was inevitably permanently recorded, and quite naturally influenced the statistician in his classification.

THE CONSEQUENCES

Most anaesthetists will be familiar with the statistical table which appears as table 41 in the Registrar-General's report under the general heading of poisoning, along with similar tables for accidents and suicide. In this table the various combinations of drugs are shown and the anaesthetic is referred to as a contributory cause. This report, of course, is a statutory one laid before Parliament each year to convey the state of the nation's health, including matters concerning public safety.

A study of these tables in retrospect provides fascinating reading, and as the first table appeared in 1908 (Registrar-General, 1908), provide a miniature history of at least the fashions in anaesthesia over 50 years. In the first of these tables there were 36 deaths so classified, the anaesthetics used being chloroform or ether, alone or in combination, and in only one was ether alone used. When compared with the latest table of 1955 (Registrar-General, 1955) in which there were 150 deaths under 62 different combinations, they appear to be of some significance. Just how significant they may be, however, depends entirely on the circumstances of each case; the only circumstances permanently recorded apart from private records are the details of the anaesthetic. When all the other circumstances are forgotten, these remain in the death certificate, the Register of Deaths and the Vital Statistics, and, of course, in any reports of the nature I have mentioned earlier. In 1956, for the first time ever, the Report of the Ministry of Health in England (M.O.H., 1954) devotes an entire section to the subject of deaths associated with anaesthesia in England. The procedure and terms used, of course, differ from those in Scotland. It is interesting, however, to note that in the year under review—1954—these deaths accounted for 1 in every 1,000 of all deaths in the population. When the same formula is applied to Scotland in the same year, the figure is 1 in 500, and in 1955, 1 in 411.

This figure in itself is sufficient argument that at least a correct interpretation of the term is made in the first place. It acquires a sinister significance, when quoted ambiguously that the expectation of dying under an anaesthetic is 1 in 1,000 as I have read in at least one authoritative textbook (Glaister, 1953). The average person interprets this as 1 per 1,000 anaesthetics, which is not the case. When stated in reference to the figures in Scotland, it is positively alarming, particularly as it is becoming progressively worse, because of the slightly falling total number of deaths from all causes and the increasing number and age incidence of the patients at risk.

Before I conclude, there is one further point worth mention. According to a memorandum from the Central Legal Office (which is not to be confused with the Crown Office), doctors in hospitals are requested to furnish reports of any deaths which are the subject of an enquiry under the Acts of 1895 and 1906 (C.L.O.1, 1955). If interpreted literally, therefore, one should furnish a report of these cases and I know that this is, in fact, being done in one particular group. Whether this is of any importance it is difficult to assess, but when one realizes that one of the reasons for making any report to the Central Legal Office is to forewarn the Department's legal advisers of possible eventual litigation (N.H.S., 1955), the implications of furnishing a report of a death under an anaesthetic to this office, I feel, cannot be ignored. This, of course, is a personal view, as indeed are all the opinions I have expressed and about which I claim no special knowledge or inside information, apart from reference to the

official instructions and documents I have mentioned.

CONCLUSIONS

I have attempted to review the legal aspects and some of the implications and consequences of the use of the terms "Death under Anaesthetic" and "Anaesthetic Death". I have done so because of the misleading impressions which may be conveyed and perpetuated by the use of these terms in circumstances where it seems unreasonable to use them. Much of this, I feel sure, could be avoided by the use of common sense in interpreting our responsibilities in this matter in the first place, and, because I fear so many of these interpretations are based on hearsay, I have taken the liberty of circulating what official instructions there are.

There remain, however, the cases which really do die under an anaesthetic. It is possible that because of the type of case at risk, in an increasing number of these the anaesthetic may be more of an incident than a cause of death. Whether or not it would be practicable to sort these out in their true perspective without minimizing the part played by the anaesthetic, may be a matter which is worthy of the attention of this Society, with a view to a possible reconsideration of the phraseology and procedure used in reporting and certifying these cases.

ACKNOWLEDGMENTS

I would like to thank Dr. Shearer and his staff at at Dundee Royal Infirmary; our own Procurator in Perth, Mr. Macmillan; the Crown Agent's Secretary, Mr. Bowen; the Chief Statistical Medical Officer of the Department of Health, Dr. McKinlay; and the statistical staff of the Register House for all their help, information and advice.

REFERENCES

B.M.J. Leader (1956a). Anaesthetic hazards in obstetrics. *Brit. med. J.*, **1**, 341.

—— (1956b). Anaesthetic deaths. *Brit. med. J.*, **2**, 868.

Brotherton, J. H. F. (1956). Royal Medical Society. November.

C.L.O.1 (1955). Central Legal Office Procedure. Regional Hospital Boards.

Glaister, J. (1953). *Medical Jurisprudence and Toxicology*, 9th ed., p. 620. London: E. and S. Livingstone, Ltd.

McKinlay, P. L. Chief Statistical Medical Officer, Department of Health for Scotland. (Personal communications.)

M.O.H. (1954). *Report of the Ministry of Health*, part 2, p. 165. H.M.S.O.

N.H.S. (1955). Legal Services for Regional Hospital Boards (Memo. S.H.M. 55/42).

Registrar General (1908). *Annual Report of the Registrar-General for Scotland*. H.M.S.O.

—— (1955). Table 41, p. 213. H.M.S.O.

EDITORIAL NOTE

This paper was shown to an English Coroner who made the following comments:

(1) There are no forms for reporting "anaesthetic deaths".

(2) In the Home Office return there is no column for "anaesthetic deaths".

(3) The usual finding is "some cause" and that the death has been accelerated by the anaesthetic, the verdict is "Death from Misadventure".

(4) In the case where the patient recovers from the anaesthetic and lives for some time, less than twenty-four hours, the anaesthetic is generally ruled out as causal.

ERRATUM

Reference: Nunn, J. F. (1957). Physiological aspects of artificial ventilation. *Brit. J. Anaesth.*, **29**, 540.

Insert "tends to" before "expands" and delete the "s" in "expands" in line 8 of the second paragraph of page 541.

CORRESPONDENCE

PARTIAL PRESSURES OF OXYGEN IN THE MAGILL ATTACHMENT

Sir,—May I be permitted to express some criticisms of the conclusions drawn by Drs. N. J. Micklem and C. S. Jones in their paper entitled "Partial Pressures of Oxygen in the Magill Attachment" (*Brit. J. Anaesth.* (1957), **29**, 491). The authors studied the oxygen partial pressures in gas samples obtained from just proximal to the reservoir balloon mount (point A) and from the facepiece (point B). They state that the samples were drawn continuously through the sampling chamber of the Pauling Oxygen Meter over a period of one minute and that the reading of the oxygen partial pressure was taken at the end of the minute of sampling.

The volume of gas in the sampling chamber of the oxygen meter at the time that the reading was made is small in relation to the tidal volume of any patient in the age grouped studied: unless this small volume is sucked unusually slowly into the Pauling apparatus, its oxygen partial pressure represents the oxygen partial pressure of the gas in the Magill system at one particular instant. The small samples taken from point B may, therefore, have been aspirated from the facepiece at any stage of the respiratory cycle—during inspiration, expiration, or during the expiratory pause. Since the authors make no comment on this, one assumes that the stage of respiration of which the samples were representative was entirely a matter of chance.

If all the facepiece samples from which readings were taken happened to be aspirated towards the end of inspiration, and if the patient's minute volume in no case exceeded the minute flow, 8 litres, of fresh gases from the machine, then it might be expected that at any instant the oxygen partial pressure at B would be equal to that at A (Mapleson (1954). *Brit. J. Anaesth.*, **26**, 323). On the other hand, if all facepiece samples were withdrawn towards the end of the expiratory phase, it would be expected that the values for the oxygen partial pressure obtained from them would be less than those obtained simultaneously from point A: the oxygen partial pressure from the facepiece samples would approach those in the patient's alveolar air. Hence, no matter how efficient the apparatus, it would always be possible to obtain low oxygen concentrations in the facepiece at certain phases of respiration.

Drs. Micklem and Jones found that the oxygen partial pressure after 20 and 30 minutes of anaesthesia was consistently lower at B than at A, and conclude that this fall in partial pressure was due to the diluent effect of exhaled nitrogen, water vapour, and carbon dioxide. They further suggested that the use of the technique described may lead to a slow accumulation of carbon doxide in the Magill attachment. Without further knowledge of the phases of the respiratory cycle at which the facepiece sampling was done, this conclusion seems unjustified.

Having made this point, I am bound to concede that the results are, nevertheless, surprising. It must be a remarkable coincidence if all the 32 samples obtained from the facepiece were withdrawn by chance during those parts of the respiratory cycle when contamination might be expected. By the rules of probability, some of the samples might reasonably be expected to have been taken during late inspiration, and therefore to show oxygen partial pressures equal to those at point A at the same instant.

It would surely have been more appropriate and the results less confusing if, instead of using the facepiece as the second sampling point, a position close to the expiratory valve in the corrugated tubing had been used. This at least would have tested Mapleson's analysis directly, without introducing the complication of the dead space in the mask, which in any case is not unique to the Magill circuit.

However, another discrepancy raises doubts of the accuracy of one or more of the measurements quoted. Samples taken from point A were led to the analysis chamber of the oxygen meter where their pressures were presumably equilibrated with the ambient atmospheric pressure. Since these samples from A contained nitrous oxide, oxygen,

and ether vapour only, the sum of the partial pressures of these three gases as they stood in the meter should have been equal to the atmospheric pressure, A. Using the usual notation for partial pressure,

$$P_{N_2O} + P_{O_2} + P_{Ether} = A$$

But the flow rates of nitrous oxide and oxygen are given as 6 litres/minute and 2 litres/minute respectively. Therefore,

$$P_{N_2O} = \frac{6}{2} P_{O_2}$$

$$= 3P_{O_2}$$

Hence, $$P_{Ether} = A - 4P_{O_2}$$

Substituting the given values of A and P_{O_2} at point A at the 20th minute for patient No. 1,

$$P_{Ether} = 771 - (4 \times 212)$$

$$= -77 \text{ mm Hg},$$

i.e. the partial pressure of ether in this case comes out to be a negative number. The same absurdity results from calculating P_{Ether} in 23 of the 32 samples obtained from point A. In the remaining 9 samples the ether partial pressure is positive.

These findings make one suspect that some fault or lack of sensitivity in the flowmeters has led to inaccuracy in the essential data, and cast grave suspicion upon the whole set of observations.

E. PAUL FITTON
Rochester, New York, U.S.A.

NOTICE OF THE ITALIAN CONGRESS

We have received further particulars of the Congress on Curare and Curarizing Drugs to be held as part of the 11th Congress of the Society of Italian Anaesthesiology at Venice from September 12 to 15, 1958.

The conference is concerned with problems associated with chemistry, pharmacology, physiology and clinical use of muscle relaxing drugs. There will be papers and short communications on muscle relaxing drugs and their antagonists. Each paper will be followed by a discussion.

Panels will be held on the following subjects:

1. Chemical and pharmacological classification and interpretation of the mechanism of action of neuromuscular blocking agents.
2. Clinical use of muscle relaxants.
3. The effect of muscle relaxants on operative mortality.

Simultaneous translations of papers, discussions and comments shall be arranged for in English, French, German and Italian.

An attractive social programme has also been arranged including visits to Lace and Embroidery Manufacturers, Venetian Glass Factories and various historical palaces. The Congress dinner will be held at the Hotel Excelsior, Lido.

Enquiries, etc., should be addressed to the address given below before March 31, 1958.

The Organizing Committee Secretary,
Ospedale al Mare,
Lido di Venezia,
ITALY.

Printed in Great Britain by JOHN SHERRATT & SON, Park Road, Altrincham

THESIA

se comes
absurdity
f the 32
emaining
positive.
hat some
eters has
and cast
observa-

TON
, *U.S.A.*

gs
at

y,
rt
be

of

or

d
s.

l,